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THE CLINICAL AND PATHOLOGICAL FEATURES OF THE DYSENTERIES.[1]

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My bowels boiled, and rested not.

JOB, XXX, 27.

I am poured out like water, and all my bones are out of joint; my heart is like wax: it is melted in the midst of my bowels.

PSALMS, XXII, 14.

GENERAL CONSIDERATIONS.

Introduction.

IT has been said, with considerable truth, that of the inventions and apparatus on which modern civilization depends, few are of greater significance than the water-closet. Without it, in cities where a million or more people may live in close proximity, the problem of keeping controlled epidemics spread by excreta would be well nigh insuperable. The elementary facts of hygiene and sanitation, painfully acquired, slowly absorbed, and now unconsciously accepted, have in themselves, by controlling these diseases, played a major part in the striking reduction of the death rate over the last hundred years, especially in children. Of the epidemics which for their origin, continuance and devastation depend on a primitive system of hygiene, few have had such importance in the home and in the field as dysentery.

War conditions impose a revolutionary way of living on those in the services. The family unit of four or five gives way to the army unit of hundreds. Garbage and fæces disposal, food protection and personal hygiene are liable to neglect. Under some conditions of service only the most elementary precautions are possible. It is not to be wondered at, therefore, that in warfare dysentery has often played a dominant role and has sometimes affected the final issue. The greater the disparity between army and civilian standards of hygiene, the higher becomes the incidence of all intestinal diseases.

It is to be remembered that bacillary dysentery is not a tropical disease only. It is ubiquitous, and whenever the complex chain of protection is broken, the disease appears.

In peace-time dysentery is no longer a serious problem in a civilized community. With babies and lunatics, however, excretory and feeding habits lend themselves to the spread of intestinal disease.

Definition.

Dysentery has been defined as a disease characterized by the passage of liquid stools in which mucus and blood appear. This was a useful definition until the laboratory methods of the last fifty years enabled us to separate this symptom-complex into a number of diseases, entirely different in their ætiology. Moreover, in some cases of dysentery, blood and mucus never appear in the stool. It is now customary to include only inflammatory conditions of the large bowel, although some small bowel diseases must be differentiated in the diagnosis.

With these considerations in mind, we therefore define the dysenteries as being composed of diseases in which diarrhœa occurs, usually with blood and mucus in the stools, owing their origin to pathogenic bacillary, protozoal and helminthic agents which cause colitis.

Without question the bacillary group is the predominant dysentery in peace and war, in the tropics or in temperate climates. Second only in importance is amœbic dysentery. These two conditions should occupy our greatest attention in any consideration of the group, as they will here.

Classification of the Dysenteries.

The diseases which will be considered are as follows:

1. BACTERIAL.
   - *Bacillus dysenteriæ* Shiga.
   - *Bacillus dysenteriæ* Schmitz.
   - *Bacillus dysenteriæ* Flexner (types I-VI).

[1] The Stawell Prize Essay, 1945.

Bacillus dysenteriæ Boyd (types I-III).
Bacillus dysenteriæ Boyd IV (P274).
Bacillus dysenteriæ D19.
Bacillus dysenteriæ Sonne.

2. PROTOZOAL.
Amœbic—
(i) *Entamœba histolytica.*
(ii) *Iodamœba butschlii* (sub judice).
Ciliate: *Balantidium coli.*
Coccidial: *Isospora hominis.*
Kala-azar.
Malaria (*Plasmodium falciparum*).

3. HELMINTHIC.
(*a*) *Bilharzia mansoni.*
(*b*) *Bilharzia japonicum.*

In this list some comment is needed on some of the inclusions and omissions.

Salmonella infections are ordinarily regarded as gastro-enteric disturbances and included as "paradysenteries". There is no evidence of large bowel involvement, and by our definition the group is excluded. In a diagnosis they must be considered, since some types cause a clinical condition indistinguishable — except by bacteriological methods—from bacillary dysentery.

The Boyd types I–IV and D19 are Flexner-like organisms, each with a specific type antigen but no group antigen, and giving the biochemical reactions of the Flexner group (Boyd[20]).

The reasons for the tentative inclusion of *Iodamœba butschlii* are found in a consideration of this amœba discussed later. The pathogenicity of this amœba is still *sub judice.*

Flagellate organisms are by some included with the dysenteries. While it is possible that *Giardia lamblia* may cause diarrhœa, the evidence for its pathogenicity is not complete. Moreover, it inhabits the small bowel only, and so finds no place in a classification within our definition.

Malaria and kala-azar, while not primarily diseases of the large bowel, may invade the colon and give rise to symptoms predominantly dysenteric. Of the others, ciliate and coccidial dysenteries, which are rare and of little importance, will be dealt with summarily.

BACILLARY DYSENTERIES.

Pathology.

Morbid Anatomy.

It is convenient and proper to group together all the species of bacillary dysentery, although in the main our knowledge of the pathology has been gained from the two most common and fatal forms—those due to *Bacillus dysenteriæ* (Shiga) and *Bacillus dysenteriæ* (Flexner). However, there is no reason to doubt that the morbid processes arising from the other milder infections are different only in degree and not in kind.

Care is needed in the interpretation of pathological changes in the bowel found at autopsy. Post-mortem changes very rapidly occur and unless the body is examined almost immediately after death, reliable conclusions on the state of the mucosa cannot be drawn.

By viewing the rectum and sigmoid colon through a sigmoidoscope at various stages of the disease, it is possible to see the earliest changes of the rectal and sigmoidal mucosa and to observe the living pathology of the disease as it progresses. All gradations, from the very mildest inflammation to a severe coagulation necrosis, may be observed. A much truer picture is thus seen than in post-mortem examinations.

Acute and Subacute Bacillary Dysentery.—The large bowel, and especially the sigmoid colon and rectum, bear the main brunt of the infection. In fatal cases the last few feet of ileum are sometimes markedly involved.

In the initial acute stage generalized hyperæmia, œdema which obscures the submucosal blood vessels, and excess of mucus are main changes. As the disease progresses, ulceration and hæmorrhages of the mucosa appear, the hyperæmia becomes more intense, mucus becomes more abundant, and swabbing leads to hæmorrhage. The ulcers originate in an extension, by coagulative necrosis, of the infection which starts in the crypts of Lieberkühn. Sometimes actual retention cysts are formed, and these break down into ulcers. In others, solitary lymphoid follicles are mainly affected. Such a development may occur in the acute, subacute, or chronic stages. The author has seen a patient with a Flexner V dysentery who illustrated well the development of ulcers from retention cysts.

He was a nineteen year old leading aircraftman of the Royal Air Force who was admitted to hospital on the day of onset. The stools showed a typical bacillary exudate, and from them *Bacillus dysenteriæ* Flexner V was isolated. The course was subacute—two to four bowel actions daily with little or no pain. On the eighth day of illness his bowel mucosa showed generalized hyperæmia and was bathed in mucopus. Organisms were still present. On the twelfth day the valves were thickened and œdematous. Scattered throughout the bowel wall were raised yellowish lumps, the size of rice grains, covered by intact epithelium (Figure I). They were surrounded by small zones of hæmorrhage. Isolated hæmorrhagic spots were also seen. The intervening mucosa was hyperæmic in patches, but the vessels could be seen. Flexner V organisms were grown from a swab at this stage. Eight days later there was still mild diarrhœa, the stools contained mucous seeds, showing a typical bacillary exudate, but were more formed, and no organisms were cultured. Sigmoidoscopy on this day showed that the small yellowish lumps had now discharged their contents and scattered ulceration was present (Figure II). The intervening mucosa was relatively normal, showing some hæmorrhages and hyperæmia, although the vessels could still be seen.

FIGURE I.

In cases fatal in the acute stage, autopsy shows the changes already described. The whole process is more severe though, and more widespread. Numerous ulcers are apparent and in parts the mucosa may be entirely stripped, the hæmorrhagic submucosa being left showing. The bowel wall is thickened and œdematous, with flakes of fibrin attached to the peritoneum in some cases. The lumen contains liquid fæces with blood and mucus.

The peritoneal cavity contains some free fluid and the mesenteric lymph glands are hyperæmic and enlarged.

FIGURE II.

Toxic changes in the viscera, spleen, liver, kidneys and heart occur, but are usually not very marked. Pericarditis may supervene.

Chronic Bacillary Dysentery.—In the last war chronic dysentery has been described as a not uncommon sequel to an acute attack. It is known to occur in native populations with a high incidence of dysentery and poor nutrition; but even before sulphaguanidine became generally used its incidence in the Middle East and New Guinea in this war was low. This question will be discussed later. Only one definite case occurred among about 1,000 cases of

dysentery seen by the author in the Middle East and New Guinea before sulphaguanidine was freely available.

This soldier gave a history of clinical dysentery starting six months before admission to hospital. He was not sent to hospital. From then on his bowel actions were never normal, often up to twelve motions daily being passed, and for the month before admission he noticed blood in the stools. Sigmoidoscopy showed ulceration very similar to that shown in Figure II, with comparatively normal intervening mucosa. *Bacillus dysenteriæ* Shiga was grown from his stool on MacConkey's medium.

A more severe form occurs having a very chronic course, severe ulceration, fibrosis of the deeper layers, and eventually colonic dysfunction.

In the recovery stages, if the loss has not been extensive, the shed epithelium is rapidly regenerated and eventually a completely normal appearance results. Deeper ulcers heal more slowly with gradual growth of epithelium, leaving a depressed area covered by bright red mucosa, which finally leaves no trace. Repair, where very extensive ulceration occurs, involves inevitably a degree of fibrosis, and may never be complete, leaving a raw, granulating surface which bleeds easily, and eventually a defunctioned colon.

The pathology of the "carrier" will be dealt with later.

Histopathology.

In the acute early stage, dysentery bacilli invade the crypts of Lieberkühn, setting up an inflammatory reaction. As a result, coagulation necrosis occurs. The histopathology of the mucosa can readily be seen in the "mucus" of stools in acute cases. These small lumps of mucus, when washed in saline solution, can usually be seen to consist of thin, flat sheets of mucosa. The microscope shows this to be epithelium in which the cells are largely replaced by polymorphonuclear cells. With additional red cells and macrophages this is the appearance of a "typical bacillary exudate". The macrophages may be feebly motile and contain fat globules and a few red cells. The importance of distinguishing them from the trophozoites of *Entamœba histolytica* is obvious.

Section through the bowel wall of patients dying in the acute stages shows shallow ulcers covered with mucus and necrotic material, and areas of coagulation necrosis. Throughout the mucosa are areas of hæmorrhage and focal necrosis. The solitary lymphoid follicles are enlarged and their cells mainly polymorphonuclear (Figure III). Sometimes retention cysts form, which may break down, leaving ulcers (Figure IV).

In the chronic stage, ulceration is deeper, although rarely penetrating the muscularis layer. The ulcers appear mainly on the bowel folds and thus are predominantly transverse. Limited areas of heaped-up granulation tissue may be seen, or this condition may be widespread, in some cases leading to the formation of pseudopolypi. Extensive fibrosis may occur, finally leaving a rigid, fibrosed large bowel.

Bacteriology.

There is not yet complete unanimity in the classification of the various types which compose the Flexner group of organisms. The question can be settled only by detailed studies of their antigenic structure, granted that pathogenicity be accepted. Boyd's classification is the most satisfactory and has been adopted here.

The large number of bacteria which make up the group has been sorted in the main by two methods, (*a*) cultural and biochemical characteristics, and (*b*) agglutination reactions.

The main biochemical reactions of the dysentery group, all of which consist of non-motile, Gram-negative coliform bacilli, are shown in Table I.

Variants of *Bacillus dysenteriæ* Flexner VI, although having an identical antigen, have diverse biochemical reactions.

The primary classification is into mannite and non-mannite fermenters. *Bacillus dysenteriæ* Shiga and *Bacillus dysenteriæ* Schmitz belong to the latter, and all of the remainder, except a few Flexner strains, belong to the former group.

The antigenic structure is complex (Topley and Wilson[2]). All the strains belonging to the Shiga group are homologous; but the mannite-fermenting group are heterologous. As well as agglutination tests, agglutination-absorption and phage-sensitivity tests have been used to separate the mannite-fermenters into a number of different types.

Toxin is produced by the dysentery organisms, in considerable amounts by *Bacillus dysenteriæ* Shiga and to a much smaller extent by the others. Whether the Shiga toxin consists of two separate toxins—one neurotropic (exotoxin), the other a toxin with affinity for the bowel (endotoxin)—is not certain. It seems more likely, on the evidence, that there is only one toxin present in the cell which, on autolysis, yields a diffusable exotoxin.

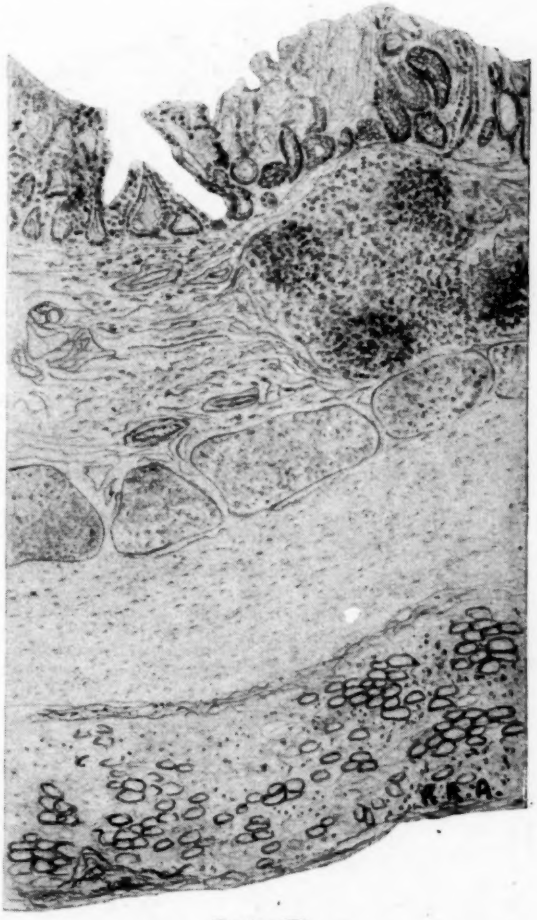

FIGURE III.

Section through caecum of a New Guinea native who died in the subacute stage three weeks after the onset of acute Flexner dysentery. (Autopsy three hours after death.) The section shows: 1. Mucosa: coagulation necrosis; ulceration commencing; polymorphonuclear and macrophage infiltration; excessive mucous secretion of intestinal cells (foamy); hæmorrhages. 2. Submucosa: solitary lymph follicles largely replaced by polymorphonuclear cells; hæmorrhages. 3. Circular muscle layer: toxic spoiling. 4. Longitudinal muscle layer: toxic spoiling. 5. Subserous coat: great increase in width with increased fat and small round-cell infiltration. 6. Peritoneum: thickened and roughened.

Clinical Pathology.

For the successful isolation of bacilli from the stools it is important to use only very recently passed specimens

for cultivation and to plate this on media using a piece of fresh mucus. This is taken from the specimen by a hooked platinum wire and washed successively in two tubes containing 5·0 millilitres of normal saline solution until free of fæcal matter. Part of this is then inoculated on the medium. After eighteen to twenty-four hours' growth, likely looking colonies are subcultured to an agar slope—a determination having first been made that the organism is non-motile—and also inoculated into the sugars. From the growth which occurs on the agar slope,

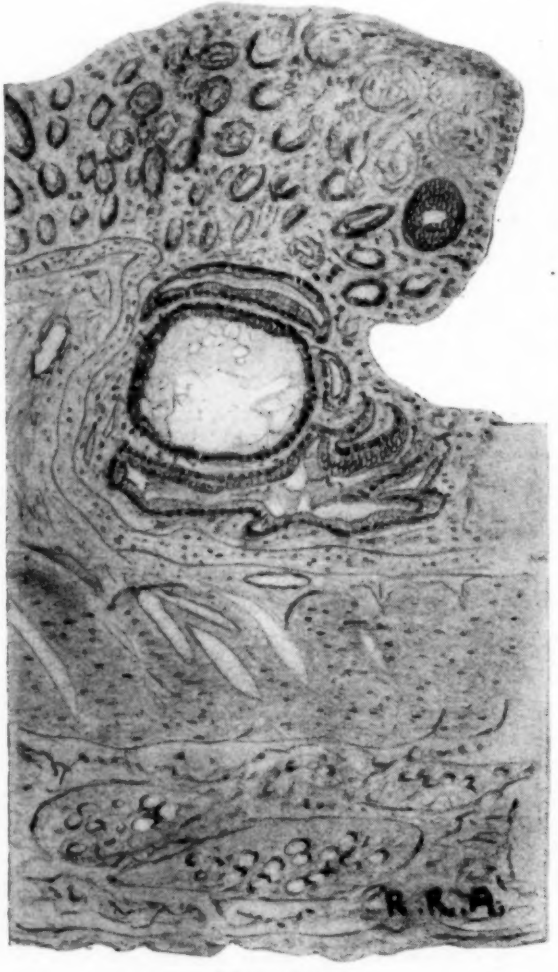

FIGURE IV.
Same case as Figure III. Section through sigmoid colon showing ulcer crater and large mucous retention cyst.

after five to six hours' incubation, an emulsion is made for slide agglutination, known antisera being used. Another piece of mucus is examined microscopically. This in the acute stages represents a portion of bowel epithelium in which the cells have been largely replaced by polymorphonuclear cells and containing mainly epithelial cells, macrophages and usually red cells. This is a "typical bacillary exudate". An "indefinite exudate" only may be found initially and is commonly seen after the acute phase is over. This consists of a mucoid secretion, embedded in which are scanty pus cells, perhaps a few red cells, and abundant intercellular mucus. All graduations of this type of exudate are seen up to a typical bacillary exudate.

The damage done to the bowel wall depends on the severity of the infection. Hence in a severe infection bacilli are numerous and a typical bacillary exudate is seen. From such a stool a high percentage of isolations will be possible. With an indefinite exudate at any stage of the disease it is far more difficult to grow the organisms. In liquid fæces with no mucus the chance of isolating dysentery organisms was small prior to the introduction of the newer selective media. In a series of 235 stools from patients in the Middle East (Hone, Keogh and Andrew[3]) the following results were obtained with the above technique and MacConkey's medium.

	Percentage of Organisms Isolated.
Typical bacillary exudate, red cells present ..	90% (83 cases)
Typical bacillary exudate, red cells absent	70% (43 cases)
Indefinite exudate	23% (53 cases)
No exudate	14% (56 cases)

There was every reason to suppose that the vast majority of patients from whom no organisms were grown were suffering from bacillary dysentery.

In convalescence, when the stool is formed, not infrequently on the scybali there are found tough mucous shreds, firmly adherent or embedded in the fæces. Microscopically they consist of closely packed squamous-like epithelial cells with a few polymorphonuclear cells and red cells varying from a few to so many that the mucus macroscopically is blood-streaked. This should not be confused with a typical bacillary exudate. Organisms are rarely present.

The organisms encountered in an epidemic are seldom limited to one type. It is the rule to find a variety of organisms with *Bacillus dysenteriæ* Shiga becoming more prominent in the tropics. Pure epidemics of Sonne dysentery, however, occur (Hailwood[4]). Rothstadt, Fenner and Baker[5] have reported an epidemic in Queensland in which half the organisms isolated in four months were *Bacillus dysenteriæ* Boyd IV (P274). In a series of 188 cases observed in the Middle East,[3] the following organisms were isolated:

Bacillus dysenteriæ Shiga	13
Bacillus dysenteriæ Schmitz	15
Bacillus dysenteriæ Flexner	148
Bacillus dysenteriæ Sonne	12

Later, monovalent serum was available, and of 101 strains the following types of the Flexner group were identified:

Bacillus dysenteriæ Flexner I	10	
Bacillus dysenteriæ Flexner II	55	
Bacillus dysenteriæ Flexner III	4	90
Bacillus dysenteriæ Flexner IV	8	
Bacillus dysenteriæ Flexner V	5	
Bacillus dysenteriæ Flexner VI	8	
Bacillus dysenteriæ Boyd I	5	
Bacillus dysenteriæ Boyd II	3	9
Bacillus dysenteriæ Boyd III	1	
Bacillus dysenteriæ Boyd IV (P274) ..		2

In a New Guinea series of 98 patients in the author's care the isolations were as follows:

Bacillus dysenteriæ Shiga	5	
Bacillus dysenteriæ Schmitz	11	
Bacillus dysenteriæ Flexner I-V	47	80
Bacillus dysenteriæ Flexner VI	33	
Bacillus dysenteriæ Boyd IV (P274) ..	2	

On occasions two types of dysentery organism may be isolated from the same patient. Probably this occurs more often than is recognized, since slide-agglutination is often performed from only one colony after subculture on an agar slope. If a number of colonies were subcultured this finding would be more frequent.

If in the mucus of the stool an exudate is seen and *Entamœba histolytica* identified, it should still be cultured, since bacillary and amœbic dysentery may coexist and each will require simultaneously different treatments.

In some severe cases, especially Shiga infections, there may be evidence of renal failure—albuminuria, granular casts, and nitrogen retention (Fairley and Boyd[6]). In a small group of 21 cases observed in New Guinea by the author, consisting of thirteen Flexner, four Schmitz, one Shiga and three clinical dysenteries, attention was focused on any evidence of renal damage. All patients were given

TABLE I.

Organism.	Medium.					
	Lactose.	Glucose.	Mannite.	Dulcite.	Sucrose.	Indole.
Bacillus dysenteriæ Shiga ..	–	A[1]	–	–	–	–
Bacillus dysenteriæ Schmitz ..	–	A	–	–	–	+
Bacillus dysenteriæ Flexner ..	–	A or AG	A	+ A or AG (late) or –	A + or –	+ or –
Bacillus dysenteriæ Sonne ..	A (late)	A	A	–	A (late)	–

[1] "A" = acid; "G" = gas.

sulphaguanidine. None was severely ill. The urine was tested on the patient's admission to hospital and thereafter two or three times during the course of the attack. In no case was albuminuria found. Blood urea estimations on the day treatment started showed a moderate rise in only five patients—60, 60, 60, 65 and 70 milligrammes *per centum*. These figures all fell to normal values within one week. The urine was hand-centrifuged and examined microscopically on the day of admission and two or three times during the attack in each case. In 10 of the 21 cases a few red blood cells were found, and in one there was a moderate number present. These cells appeared an average of two days after the onset. In only one case were red cells seen at the conclusion of treatment, and then only a few were found. In a series of 50 patients with general diseases—mainly malaria—a few red cells were seen in the urine of 11. These observations were made in a distressingly hot and humid climate, where urinary output is low and the urine very concentrated. One can say, therefore, that in the average case of bacillary dysentery there is seldom any evidence of renal damage.

Examination of the blood shows, in most cases, a moderate leucocytosis of the polymorphonuclear type. In fact, where malaria is common and blood films are examined from all patients, an obvious leucocytosis may be seen and is not infrequently caused by dysentery, which may be suspected, in the presence of abdominal pain, before diarrhœa has started and the patient is still in the "pyrexia of unknown origin" category.

Serum from the patient may show a rise in antibody against the infecting organism; but the rise is inconstant and of no value in diagnosis.

Clinical Features.

Epidemiology.

Epidemics of bacillary dysentery are initiated and maintained by the ingestion of food or water infected by dysentery organisms. Contamination takes place by flies, infected hands or careless sewage disposal. Carriers of the disease maintain the infection in interepidemic periods. They may be divided into three main groups:

1. Those incompletely recovered from an overt attack, (*a*) who have never been treated, (*b*) who have been treated but discharged from hospital still excreting organisms, (*c*) who have relapsed.

2. Those who have clinically recovered from an overt attack of dysentery and without signs or symptoms, local or general, continue to pass dysentery organisms in the stools.

3. Those who become infected and, without signs or symptoms at any time, pass bacilli in their stools.

Possibly all these groups play their part in an epidemic. However, those in group 3 are extremely few, those in group 2 uncommon, while those in group 1 are the most numerous, the most dangerous and the most easily discovered.

In a series of 178 hospital patients observed in the Middle East (Keogh, Hone and Andrew[63]), in whose stools dysentery organisms were identified by the use of MacConkey's medium, there were only six persistent carriers. They carried two Shiga, two Schmitz and two Flexner IV infections. None was passing normal stools. None had been given sulphaguanidine, which was not then available. This low incidence of 3·5% is in general agreement with other workers' findings.

Fletcher and Mackinnon (1919)[7] found that of 935 patients with bacillary dysentery, 2·78% became persistent carriers (that is, for over three months from the onset).

It has been maintained that the test for cure was the regular passage of formed normal stools. Boyd in India, with many thousands of normal stools from healthy subjects, was never able to isolate dysentery bacilli. In the last few years, with the use of more sensitive media, such as desoxycholate-citrate and S-S media, it has been found that, in varying proportions of cases, dysentery bacilli can be cultured from stools which are normal macroscopically and microscopically. However, this seldom occurs when all clinical tests of cure—including, most importantly, sigmoidoscopy—have been fulfilled.

The epidemiological significance of these recent findings is not yet clear. A few such patients (groups 2 and 3) in a position of vantage (cooks and food handlers) may be the prime movers of an epidemic. But it seems unlikely that they can be of much importance in the maintenance of infection at epidemic levels. A healthy subject passing one formed normal stool daily, on the surface of which a few organisms can be cultivated by very refined means, must weigh very little in the balance against those in group 1, mentioned above, who are the main "carriers" of the disease. The chances of flies becoming infected or hands contaminated by the healthy subject must be very small. This question is not merely of academic interest, and the significance of these conclusions and their importance in management will be dealt with in the sections on criteria of cure and control.

The author's experience of most epidemics is that contamination of food by infected flies is the common means of spread. This is in conformity with many observations by numerous workers. However, hand-to-mouth contamination does occur.

In the Owen Stanley Mountains of New Guinea (1942) troops were initially moving through virgin jungle. For some weeks, and before the trail was fouled, extremely few flies were seen, although the dysentery rate was alarmingly high. These troops had returned from the Middle East six months before this, and no doubt there were dysentery carriers amongst them. Privation was extreme on the Kokoda trail and the men were wet most of the time. Defæcation could not be other than primitive, and hand contamination the rule. In these circumstances it is not to be wondered at that, with the equally primitive messing arrangements—a tin of "bully" going from hand to hand—dysentery should break out.

Another example of an epidemic, caused by a carrier contaminating food, will be found in the section dealing with dysentery in the army.

Water contamination is uncommon, but does occur. The author has had five victims exemplifying this in his care in New Guinea.

Five men with diarrhœa, from one unit, were admitted on one day. They had arrived from Australia four days previously. In the space of five hours all had developed diarrhœa. From two men *Bacillus dysenteriæ* Flexner (VI) organisms were isolated from typical bacillary exudates. Of the other three, one showed an indefinite exudate, one had fluid motions with no exudate, and one had soft motions with no exudate, and from none of these were

organisms isolated. Subsequently it was learned that fifteen other men in the same unit had mild diarrhœa on the same day but did not report sick. Major A. A. Ferris succeeded in isolating an inagglutinable[1] dysentery organism from their water tank, which was not being properly chlorinated.

This small outbreak illustrates well the protean characteristics of the disease, varying from a mild transient diarrhœa in some patients to a sharp attack with blood and mucus in the stools.

Incubation Period.

The incubation period is considered generally to be two to seven days. However, a patient under the author's care in New Guinea illustrates that this can be considerably shorter.

The patient was a staff sergeant from a mobile laboratory. At the time of his attack, there was a considerable number of inagglutinable mannite-fermenting dysentery organisms isolated from patients with clinical dysentery. It was questioned if these were pathogenic. He determined, unknown to anyone else, to try this on himself. From a patient with dysentery, organisms with the above characteristics had been isolated and subcultured on an agar slope for eighteen hours. To this he added 2·0 millilitres of normal saline solution. He then pipetted 0·1 millilitre of the emulsion, mixed it with water, and swallowed it at 1700 hours. At 0300 hours next day, that is, ten hours later, he awoke with severe abdominal colic. A rigor followed and he developed a headache. His bowels were open a quarter of an hour later. Sulphaguanidine was commenced three hours later. His course was that of a moderately severe attack of dysentery with a typical bacillary exudate. An inagglutinable mannite-fermenter dysentery organism was isolated from his stool. A few weeks after his recovery, monovalent Flexner antiserum arrived, and when his organism and the original culture were retested they were both found to be *Bacillus dysenteriæ* Flexner VI. Apparently the titre of the Flexner VI contained in the polyvalent serum number 2 (Flexner IV, V and VI) was at fault. The pathogenicity was thus proved.

The incubation period in this case—ten hours—followed an enormous dose of organisms, and represents probably about the shortest period possible. In nature, longer incubation periods, which are usual, perhaps result from a smaller dose of organisms.

Symptomatology.

The onset is acute with abdominal colic in attacks succeeded by generalized abdominal discomfort. Within a short period in most—although a day may elapse—this is followed by urgent diarrhœa, which gives only temporary relief from pain. There may be a short-lived rigor at the onset, headache is common, and generalized aches and pains in the muscles may occur. The stools become fluid, and blood, mucus and pus appear in the more severe cases. Considerable quantities of blood are passed in most Shiga infections. Muscular cramps occur only in severe cases.

In the Middle East, 62 cases of proven bacillary dysentery comprising all types (Shiga, Flexner, Sonne and Schmitz) were investigated in detail (Hone, Keogh and Andrew[(2)]). A special *pro forma* was used, and a detailed history and follow-up were kept to elicit exact information. Sulphaguanidine was not available at the time—except for severe Shiga infections—and all were treated by diet and saline purgatives.

Nausea and Vomiting.—Half the patients (29 of 60) complained of nausea preceding or accompanying the onset of diarrhœa, and one-third (19 of 59) vomited. The vomiting was never severe or persistent. Twelve vomited once, three vomited twice, and the remaining four vomited several times. In four cases vomiting preceded the onset of abdominal pain, in eleven it accompanied the onset, and in four it appeared later.

Headache.—About two-thirds of the patients (36 of 59) complained of headache. Four patients complained of headache twelve or more hours before the onset, and in twelve cases the headache preceded the onset by a few hours.

Malaise, Aches and Pains.—About half the patients (27 of 53) complained of aches and pains in the muscles and joints. In eleven cases these symptoms preceded the onset of diarrhœa, in eleven they accompanied the onset, and in six they developed later.

Pyrexia.—The great majority of patients were pyrexial on admission to hospital. The fever was usually of moderate degree and rarely lasted more than two or three days.

Prodromal Symptoms.—Taking all the above-mentioned symptoms in combination, about one-third of the patients (17 of 53) had some prodromata, and in nine cases these preceded the onset of diarrhœa by twelve or more hours. There was no correlation between the presence or severity of prodromal symptoms and the severity of the attack.

Abdominal Pain, Tenderness and Tenesmus.—In only three out of the sixty-two cases was abdominal colic absent, and in two of these the patients complained of tenesmus. Initially, constant abdominal pain forms a continuous background to the attacks of colic, which are usually followed by urgent defæcation, and the pain gradually increases in severity over the next day or two.

Tenesmus was recorded in about a quarter of the cases (8 of 35).

Abdominal tenderness was found in most cases (52 of 61). It was commonest in the right and left iliac fossæ, next commonest in the upper part of the abdomen, and infrequent in the mid-abdominal region. Tenderness was usually somewhat diffuse but seldom generalized.

The recorded frequencies of the various sites were as follows:

Upper part of the abdomen (42)—	
Right hypochondrium	16
Epigastrium	10
Left hypochondrium	16
Mid-abdomen (21)—	
Right lumbar region	8
Umbilical region	5
Left lumbar region	8
Lower part of the abdomen (88)—	
Right iliac fossa	33
Hypogastric area	22
Left iliac fossa	33

In most cases the tenderness rapidly disappeared; in about half it persisted a few days, usually settling into the iliac fossæ. Persisting tenderness was twice as frequent in the left iliac fossa as in the right iliac fossa (14 : 7).

Diarrhœa.—The onset of diarrhœa was usually explosive. It was uncommon for mild diarrhœa to precede the attack. More than half the patients (30 of 53) had the maximum number of stools on the first day.

First day	30 patients
Second day	12 patients
Third day	6 patients
Fourth day	2 patients
Fifth day	2 patients
Ninth day	1 patient

Correlation was found between the severity of the attack, as judged by the number of bowel actions, and the duration of the disease. The scatter diagram (Figure V) shows that the patients fell roughly into two groups: those whose bowel actions in any one day did not exceed 15, and those who passed more than 15 bowel actions on one day. The former had diarrhœa for three to six days, and the latter, diarrhœa for six to sixteen days. Diarrhœa was considered to have ceased, in these calculations, when the number of stools fell permanently to three or less daily. In only four of 62 patients did diarrhœa persist for longer than sixteen days. These are included in the series of 188 patients mentioned earlier.

The main symptoms in the initial stages of the attack in the above series may be compared with another series of 50 patients in the author's care in New Guinea. This was

[1] It is not absolutely certain from this that the cause of the epidemic was isolated. However, the Gram-negative coliform organisms isolated were non-motile, and biochemically and culturally belonged to the Flexner group. Possibly dissociation—as may happen with cholera growing in water—had led to a change in antigenic structure. The epidemic stopped with efficient chlorination.

a specially selected group which will be mentioned later, and included by deliberate selection those rather more severely affected than those in the Middle East group. The infections were: Flexner, 42; Schmitz, 7; P274, 1. The frequency of symptoms when compared is seen in Table II.

So far only the average moderate attack of dysentery has been described. In the section dealing with epidemiology an example was given of the protean features of an epidemic, which needs emphasis. Some cases are so mild that the patients never reach hospital. Others may be extremely severe, especially Shiga infections, and death may occur within two days from the onset. The onset in these cases is usually acute (but may be insidious), and vomiting, collapse and dehydration rapidly appear. The temperature is subnormal, the facies pinched and anxious, the extremities cold, and the pulse rapid and weak.

TABLE II.

Symptom.	Middle East Series.		New Guinea Series.	
	Number.	Percentage.	Number.	Percentage.
Colic	59 of 62	95	49 of 50	98
Headache	36 of 59	63	29 of 50	58
Vomiting	19 of 59	32	9 of 50	18

Course of the Disease.

It has been pointed out that the presence of mild prodromal features is no indication of the course of the disease, which can be best judged by the number of bowel actions daily in the first few days of the attack (Figure V).

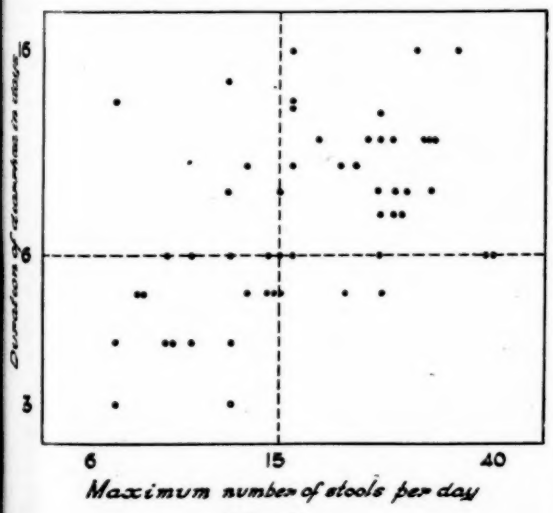

FIGURE V.

Scatter diagram showing correlation between number of stools daily and the duration of the disease.

The progress of an attack of moderate severity, if no specific treatment is given, is usually one of steady improvement clinically after the first two or three days. At the height of the disease there is fever of a moderate degree and a degree of tachycardia sometimes out of proportion to the temperature. Anorexia is fairly marked, and abdominal pain is more or less constant, with exacerbations of lower abdominal colic, and sometimes accompanied by tenesmus. The desire for defæcation is urgent, but frequently only small stools are passed with but temporary relief of the pain. At first the stool is watery and fæcal. Soon small mucous "seeds" appear, to be followed, in the typical case, by blood and mucus which is like egg white and sticky. There may be no fæcal matter at this stage. Later the mucus may be seen streaked with pus in severe cases. Headache is common, although not very severe. After a few days colic becomes less frequent, the abdominal discomfort fades and the appetite returns. Tenderness becomes less marked and settles mainly in the iliac fossæ—twice as commonly in the left as in the right. With this improvement the stools decrease in number, their character alters, blood and mucus becoming less and fæcal material more abundant. The average times for these changes may be seen in Table III, comprising a series of 25 cases observed in New Guinea. They represent the less severe cases that were occurring at the time. No drugs were used in their treatment.

TABLE III.

Observation in Twenty-five Cases.	Average.	Range.
Average days before admission ..	1·7	1– 5
Maximum bowel actions on any one day	9	5–15
Bowel actions daily before admission	7·3	2–13
Days of pain after admission ..	3·3	0–10
Days before bowel actions two or less..	4·8	1– 7
Days before cured	7·8	4–20
Days in hospital	16	11–31

There were 22 Flexner, two Schmitz and one P274 dysenteries in this group. These figures will later be considered with a parallel series of 25 cases in which sulphaguanidine treatment was adopted.

Shiga dysentery generally runs a more severe course, although again quite mild cases may be seen. The diarrhœa is more severe, pain usually is worse and large amounts of blood are commonly seen in the stool. The attack takes longer to resolve, and, untreated, offers a greater threat to life.

The author knows of no series large enough to be valid, giving a statistical comparison in the symptomatology of the four main types of dysentery bacilli. Such a review was intended in the group of Middle East cases mentioned. However, the numbers were too small for analysis. Generally the Shiga group is most severe: the Flexner group, including Boyd's types, is mild to severe; and the Sonne and Schmitz dysenteries are usually mild. It is impossible to make an accurate diagnosis of these types clinically in the individual case.

Sequelæ and Complications.

The acute stage, in the vast majority of cases, rapidly subsides, and the patient makes a complete recovery. This is true of the average well-nourished Australian or English subject. However, in a few cases, and not infrequently with anæmic, ill-nourished coloured races, a subacute or chronic stage may supervene.

In patients whose diarrhœa persists for several weeks, and whose stools remain cellular and abnormal, it is commonly found that changes in the bowel wall, as seen by the sigmoidoscope, have developed.

The bowel wall may appear uniformly pink, with œdematous mucosa and obliteration of the superficial vessels. Mucus is often in excess of normal and on swabbing of the mucosa, which has a velvety appearance and bleeds easily, organisms can usually be recovered from the swab.

Against this background ulcers may or may not be present. They may be small or large. Only one may be seen in some cases. In others they are numerous and most commonly found on the valves of Houston. They may appear as shallow erosions or as fairly deep ulcers.

A subacute case has already been illustrated (Figures I and II), in which the ulceration was watched from the stage of yellowish retention cysts to the formation of ulcers. The intervening mucosa was relatively normal and gave a picture very similar to amœbic ulceration. Healing

may occur gradually at this stage, or the disease may continue until a chronic condition exists.

Rogers[8] states that the main incidence of chronic inflammatory changes in the great majority of cases is confined to the lower half or two-thirds of the large bowel.

Two main types occur: (i) The walls become thickened and contracted, and the lumen may be tubular in shape. On the surface is red granulation tissue which bleeds easily. The condition may be mistaken for chronic ulcerative colitis, and in fact it is possible that some cases of chronic ulcerative colitis have their origin in this way, a view which Hurst strongly supports. (ii) The mucosa shows numerous ulcers, without fibrosis of the wall, and relatively normal mucosa. Such a case was described under the heading of "chronic bacillary dysentery".

In not a few constitutionally inadequate soldiers, with or without an overt neurosis, diarrhœa may continue for many weeks or may reappear at frequent intervals accompanied by abdominal pain or colic. Examination of the abdomen often reveals a firm, contracted, slightly tender sigmoid colon. The stools are watery, but contain no abnormal constituents other than undigested food. Sigmoidoscopy reveals no pathological change other than slight hyperæmia in some cases. X-ray examination after a barium enema reveals a normal colon. These patients constitute a very difficult problem and usually belong to the province of psychotherapy. They recover in time, but rarely while in a position of much stress, for example, as soldiers in the field.

They are included by some writers in a group called "post-dysenteric colonic irritability". The author doubts the reality of this syndrome. With normal findings of stool and bowel mucosa the diagnosis is much more accurately one of psychoneurosis. The question is one of importance. On the one hand, if it is believed that an organic condition exists, attention will be focused on the bowels. On the other hand, when the condition is believed to be entirely psychological, every endeavour will be made to undo the morbid intestinal fixation which these patients acquire. The "irritability" is usually in the brain, not in the bowel.

The complications which occur most frequently with Shiga dysentery have been reviewed by Fairley and Boyd[6] and are here reproduced:

(A) INTESTINAL AND ABDOMINAL.

(1) Intestinal hæmorrhage.
(2) Intestinal perforation with peritonitis.
(3) Chronic peritonitis with localized or generalized effusion of peritoneal fluid.
(4) Pneumoperitoneum.
(5) Portal pyæmia and multiple abscess of the liver.
(6) Hæmorrhoids which may become thrombosed.
(7) Rectal prolapse.

(B) SYSTEMIC.

(1) Peripheral circulatory failure.
(2) Renal failure.
(3) Toxic arthritis.
(4) Peripheral neuritis.
(5) Conjunctivitis and iritis.
(6) Pneumonia.
(7) Parotitis.
(8) Petechial and purpuric rash.

These complications may occur with other types of bacillary dysentery, but it should be emphasized that most of them are very rare. They were collected from many thousands of cases. The only ones observed by the author in about 1,500 cases of all types have been numbers (A) 6 and 7; (B) 1 (2 cases), 3, 4 and 5. In addition, one patient has been seen with pericarditis, probably a complication of dysentery. He was desperately ill, but finally recovered. The bowel infection was moderate—a typical exudate but no organisms isolated—and the pericarditis developed one week after the onset.

Prognosis.

The mortality without specific treatment varies considerably in different epidemics. It is highest in debilitated populations living in conditions of inadequate nutrition, squalor and filth, and may be as high as 20% to 30%. Shiga dysentery is more fatal than the other groups.

In healthy adults the prognosis, even without specific treatment, is excellent and full recovery in a short period the rule. A few infections become chronic and a very small percentage of patients die.

With the advent of sulphonamides in the treatment of bacillary dysentery, the prognosis both for early recovery and for mortality has improved considerably. Given that the patient is treated reasonably early in the disease, recovery is rapid and death extremely rare.

Among 10,000 cases of dysentery in the Australian Military Forces in New Guinea, after the advent of sulphaguanidine, there were only two deaths (Fairley[9]).

In the Middle East, 1940-1942, the Australian Imperial Force had 3,806 cases of dysentery and five deaths (0·13%). Sulphaguanidine was fairly generally available late in 1940, and earlier for severe cases.

Diagnosis and Differential Diagnosis.

Diagnosis.—Many of the points in diagnosis have already been dealt with. It is proposed here briefly to summarize the various procedures necessary for an exact diagnosis.

While an epidemic of dysentery can be controlled and the great majority of patients effectively treated without the full services of a laboratory—that is, by examining the stools microscopically and macroscopically and by using the sigmoidoscope—nevertheless it is advisable whenever possible to bring into play all the resources available. Only then can an exact diagnosis be made. The physician and hygienist must always remain uneasy until bacteriological control is instituted.

For a diagnosis, consideration must be given to the three main groups into which these patients fall: (*a*) acute bacillary dysentery, (*b*) subacute or chronic bacillary dysentery, (*c*) carriers. In the army these groups broadly present as the "no duties", the "light duties" and the "full duty" dysenteries respectively.

1. Acute Dysentery. The history is nearly always of abdominal colic shortly followed by urgent diarrhœa. There is usually some constitutional disturbance preceding or accompanying the onset. The patient may notice blood and mucus. On examination early in the attack, fever and abdominal tenderness are present, usually in the lower part of the abdomen.

The next step is to examine the stools. The specimen should be absolutely fresh. A liquid stool with blood and mucus is characteristic of dysentery. Mucus from the stool is carefully selected and sent immediately to the laboratory for microscopic examination and culture. Should the patient be acutely ill, perhaps admitted to hospital at night, and no stool be available, treatment should not be withheld if a clinical diagnosis of dysentery has been made. It has been found that a few doses of sulphaguanidine do not prejudice the chances of isolating the organism (Fortune and Ferris[10]). Some authorities recommend routine rectal swabbing and immediate culture, since several hours may elapse before a stool is available. This has much to commend it, especially as the percentage of positive isolations is perceptibly higher than with plating from the stool. The diagnosis should not rest on this alone. Microscopy from the fresh stool is always necessary to exclude amœbic infection.

The author has seen a patient, with a history of recurrent diarrhœa for three years, admitted to hospital with an acute attack of dysentery starting four days before. Fifteen bowel actions occurred in four hours on the third day. Sigmoidoscopy showed only excess mucus. A swab was taken and *Bacillus dysenteriæ* Schmitz was cultured. The stools were examined next day and a few iodamœbæ were seen. A week later (after sulphaguanidine) the stools were again examined and *Entamœba histolytica* cysts were found.

Further details of laboratory diagnosis have been described earlier.

Sigmoidoscopy in the acute stages is very painful, and, moreover, unnecessary. A planned investigation may justify this procedure in some cases, but with a primary acute attack of dysentery all the information necessary for diagnosis and treatment can be gained without its use.

The appearances in the acute stage have already been described.

The Appendix gives a brief account of the essential points in sigmoidoscopic examinations.

2. Subacute and Chronic Dysentery. In these cases the acute phase is over, but diarrhœa, with the passage of abnormal stools, has continued for weeks or months continuously or in attacks with remissions. Indigestion, anorexia and colic are common, and abdominal tenderness is usual. There may be considerable loss of weight. There may also be evidence of nutritional deficiency. (The patient with chronic Shiga dysentery mentioned under the heading of chronic bacillary dysentery, had achlorhydria, angular stomatitis and a smooth atrophic tongue. These signs all disappeared with treatment.) In the later stages a condition of cachexia with colonic dysfunction exists.

In these cases sigmoidoscopy is the most important single aid to diagnosis. The appearances of the bowel wall have already been described. Curettage of the ulcers will usually lead to isolation of dysentery organisms, when inoculation from the fæces has failed.

The fæces should be examined microscopically and cultured as in the acute case. Moreover, one negative finding is not sufficient. The author has seen a case of subacute Shiga dysentery, with ulceration, in which the organism was grown only on the third attempt at culture. The organisms were recovered from the patient quoted earlier only after four attempts had failed.

In some cases in which the diagnosis is in doubt a barium enema should be given. Ulcerative colitis, diverticulitis, and carcinoma of the colon can be excluded in most cases by this means.

3. Carriers. The three groups which make up the carriers of bacillary dysentery have already been named. In the individual case the diagnostic procedures are those used for the subacute and chronic stages, with sigmoidoscopy, swabbing and direct plating as the most valuable means of detection. In mass surveys, however, rectal swabbing and cultures are the most convenient and successful methods for their detection. In a later section reference is made to a cruder method adapted to the field, where laboratory assistance is limited, which may be useful in the elimination of the dangerous carriers.

Differential Diagnosis.—Under the heading of differential diagnosis several conditions must be considered.

1. Amœbic dysentery and ulcerative colitis. The main condition to exclude is amœbic dysentery. It has been pointed out that more than one microscopic examination of the fæces may be necessary to find the amœbæ. Moreover, an exudate in association with trophozoites or cystic forms may have its origin in a double infection. A culture therefore should always be grown. By attention to the points already enumerated, with particular emphasis on the examination of absolutely fresh material, acute attacks of amœbic dysentery should not be missed. Clinical differentiation cannot be certain. In the chronic form, sigmoidoscopy and sometimes curetting of any ulcers present may be necessary before amœbæ are found. With ulcerative colitis the diagnosis may be very difficult in the acute stage. In some cases the onset is sudden, the symptoms are the same as in bacillary dysentery, and cytodiagnosis is of no help, since a typical bacillary exudate is common to both. The repeated failure to grow organisms and, above all, the failure of response to sulphaguanidine are important points. The subsequent course of ulcerative colitis is different. The disease may be rapidly fatal or characterized by repeated exacerbations and remissions of a mild or severe nature. Ulceration develops, fibrosis and narrowing occur, and ultimately the colon loses its haustrations and is converted into a thickened straight channel which gives a unique radiological picture with barium. Polyposis is usual in the terminal stages. It is obvious how difficult the diagnosis may be from chronic bacillary or amœbic dysentery in patients exposed to these diseases. Only by enlisting all the diagnostic aids possible — cytodiagnosis, culture, sigmoidoscopy, X rays and the therapeutic response to sulphaguanidine or emetine—will some cases be accurately diagnosed.

2. Salmonella Infections. Some strains will cause an attack of diarrhœa clinically indistinguishable from that of acute bacillary dysentery. The symptomatology may be the same and the stool may show a typical or an indefinite bacillary exudate. Usually the outbreak is fiercer than with bacillary dysentery, and the onset in the majority of those attacked occurs within a few hours of the meal which has conveyed the infection. It should be remembered that dysentery may sometimes occur as an epidemic, similar to food poisoning. The author has seen several examples of this, one of which has been mentioned.

3. Protozoal and Helminthic Dysentery. Protozoal and helminthic dysentery will be dealt with in their appropriate sections.

4. Acute appendicitis. In the early acute stages of dysentery the symptoms may closely imitate an acute attack of appendicitis. After the first day there should be little difficulty, but when the patient is seen soon after the onset, especially with prodromata which last for hours or even a day before the diarrhœa is acute, it may not be easy to distinguish the condition. Some cases of appendicitis start with pain and diarrhœa, and these are the ones in which considerable judgement is necessary. The most helpful points in the author's experience are the type of pain and the site of tenderness. Pain in acute appendicitis is nearly always more severe and constant than with dysentery, tenderness is not so diffuse and it is mainly in the right iliac fossa. There may be rectal tenderness on the right side, with appendicitis. Rovsing's sign is of no help. If diarrhœa has not commenced, or if only one or two stools have been passed, which are not available for inspection, a rectal swab may be of considerable help. The finding of a typical bacillary exudate will almost certainly rule out appendicitis. The two conditions may coexist and persistence of pain and tenderness in the right iliac fossa in spite of sulphaguanidine should arouse suspicion (Rose[1]).

5. Uncommon Conditions. Diverticulitis, carcinoma of the colon, tuberculosis and syphilis of the colon, and actinomycosis may need to be considered in cases of chronic or recurrent dysentery.

Management and Treatment.

In the last three or four years the treatment of bacillary dysentery has been revolutionized by the introduction of the sulphonamide drugs. There seems little point in discussing the value of saline purgatives, kaolin, antidysenteric serum, bacteriophage, intestinal disinfectants, vaccines *et cetera*. Certain general principles of treatment have survived from the presulphonamide era, but little else. There appear to be very few cases of bacillary dysentery which are resistant to specific treatment, although occasional strains are reported which do not yield readily to treatment. In the vast majority the results are dramatic, rapid and permanent.

1. *Ward Management.*—It is of the utmost importance to ensure that cross-infection in the ward does not occur and that the dysentery ward of a hospital does not act as a dangerous focus of dissemination to other wards. To this end a satisfactory scheme was devised at the 2nd/2nd Australian General Hospital by Lieutenant-Colonel E. Britten Jones. The following account is taken verbatim from a paper describing the methods used (Hone, Keogh and Andrew[2]).

> The two dysentery wards were sited as near as possible to the laboratory. They were wire-screened against flies. The annexes, situated in the centre of each ward, were modified, so that an enclosed wire-screened room was provided in the rear. In this room a large wire-screened cupboard was built to hold the bed pans containing stools awaiting inspection by the medical officer. The used pans were thus doubly shielded from flies. An incinerator was built within the room so that fæces could be destroyed without leaving the dysentery ward. The fæces were mixed with sawdust and burnt in the incinerator; this procedure gave rise to no objectionable smell. Two coppers were kept boiling beside the incinerator, in which bed pans were sterilized before being returned to wire-screened cupboards in the wards.

days, but only rarely is a purgative needed.) The improvement in the patient's general condition is much more rapid with the drug and is reflected not only in the bed-pan, but also in the patient's appearance.

Occasional patients are seen—usually neglected patients—with severe and persisting vomiting which prevents an adequate concentration of sulphaguanidine being achieved in the bowel for controlling the infection. For these a soluble sulphonamide (sodium sulphapyridine) should be given. Four grammes given intramuscularly twice a day for two days will usually control the infection and vomiting and allow sulphaguanidine to be given. Care should be taken with these patients, who are usually dehydrated and passing highly concentrated urine, that the reaction of the urine is kept alkaline to minimize the chance of sulphonamide deposition in the renal tract. Intravenous sodium lactate therapy may be necessary to achieve this.

B. Subacute and Chronic Bacillary Dysentery. The adequately treated acute bacillary form nowadays very rarely goes on to a subacute or chronic stage. Not infrequently a patient is encountered who gives a history of acute bacillary dysentery, months or several years before, and complains of recurrent bouts of diarrhœa some weeks or months apart. The vast majority of bouts are functional in origin. The less attention paid to the intestinal complaints of these patients, the better. One check, including clinical and sigmoidoscopic examination and several stool examinations, should suffice unless amœbiasis is suspected. The patients should be treated as for other psychosomatic upsets.

Chronic and subacute bacillary dysentery responds well to sulphaguanidine. The speed with which lesions will heal is astonishing. The leading aircraftman with subacute Flexner V dysentery, mentioned earlier, started sulphaguanidine on the day Figure II was drawn (twentieth day of illness). Sigmoidoscopy five days later, after 90 grammes of sulphaguanidine had been given, showed complete healing of the ulcers, which could be located only by slightly hyperæmic, depressed areas covered with epithelium which did not bleed on swabbing. Thirteen days from the start of treatment the bowel wall was normal in every way and the patient cured.

The soldier with chronic Shiga dysentery quoted earlier, with similar ulceration, was also given sulphaguanidine. Six days later sigmoidoscopy showed that the ulcers were nearly all healed. Four days after this the only evidences of inflammation were depressed areas, as in the case above, marking the site of previous ulceration. He was given 218 grammes of sulphaguanidine over eleven days.

It is worth while giving a soluble sulphonamide as well—for example, sulphamerazine—in some resistant cases, in order to raise the blood concentration to fairly high levels in the hope that any infection of the bowel wall is more adequately attacked.

Sulphaguanidine in chronic cases should be tried in doses larger than those advocated for the acute attack and given for a longer period, up to fourteen days. The diet should be full, well balanced and residue-free. Added vitamins may be required. Achlorhydria may be present and dyspepsia in these cases will diminish with the addition of diluted hydrochloric acid. A firm, optimistic approach is necessary. Grafted on many of these patients is a morbid introspection, sometimes obsessional, about the bowel functions.

Various intestinal drugs, given as retention enemata, have been used in the past. If sulphonamides fail, there seems little prospect of eusol, "Albargen", silver nitrate *et cetera* succeeding.

In severe advanced cases surgery may be necessary. Appendicostomy and caecostomy have been performed in the past with some success. This should very rarely be necessary now.

Criteria of Cure.

The criteria adopted in the series of comparative treatment (Table IV) have been: (i) The patient looking and feeling well with a good appetite, and without abdominal pain or tenderness. (ii) Cessation of diarrhœa—a normal number of stools for the individual daily. (iii) Normal stools on macroscopic and microscopic examination. (iv) Normal sigmoidoscopic appearances (this examination in selected cases). (v) Absence of dysentery organisms in cultures of stools on sensitive media in selected cases (cooks, food handlers, nurses, nursing orderlies).

In all cases criteria (i) to (iii) should be fulfilled. It is doubtful if sigmoidoscopy is always necessary. If the patient is well and the stools appear normal over a period of several days, and there is no mucus or exudate, the chance of an unrecognized, silent, subacute stage is remote. Before sulphaguanidine was introduced it was rare to find any lesion of the bowel wall if the criteria (i) to (iii) were fulfilled. (In this period, in the author's cases, nearly all patients underwent sigmoidoscopy before being discharged.) Since sulphaguanidine has become available, the chances are reduced still further. Only in cases in which the criteria (i) to (iii) are not fulfilled, and in cases of recurrent, subacute, chronic or carrier dysentery, is sigmoidoscopy necessary. It should be done at five to seven day intervals until the bowel wall is seen to be normal.

It is also doubtful if criterion (v) need be fulfilled in the average case. The author has given reasons for assuming that the carrier state is not common or dangerous when the main three criteria of cure are fulfilled. Undoubtedly a small proportion of clinically cured patients will pass organisms, which can be detected only by very sensitive media after rectal swabbing or swabbing through the sigmoidoscope. Fortune and Ferris,[40] in New Guinea, found that, after an acute attack of Flexner dysentery treated with sulphaguanidine, only 4% of patients (seven of 179) became carriers. They used desoxycholate-citrate for isolation. Only three (1·2%) passed positive stools thirty days after the initial treatment started. The epidemiological danger of these patients is questionable so long as they have nothing to do with the distribution, preparation, cooking or serving of food. Only in these cases is it necessary to make a final bacteriological clearance, and three successive swabs are advisable. The stay in hospital is reduced, and much time and material are saved in the ward and the laboratory by eliminating unnecessary control.

Infantile Diarrhœa.

Infective diarrhœa, from whatever cause, has always been the source of much illness and has resulted in a considerable mortality in infants. The ætiological agents are complex, but there is no doubt that, in some outbreaks, bacillary dysentery—usually due to *Bacillus dysenteriæ* Flexner or *Bacillus dysenteriæ Sonne*—is the cause.

The gross pathology of dysentery in children is the same as that of adults, although the clinical picture and the course often show considerable differences. It is proposed here briefly to consider the pathological and clinical features, emphasizing these differences.

Epidemiology.

The conditions of poor sanitation which cause epidemics of bacillary dysentery in adults apply equally to the disease in children. However, infection by milk is the main source of infantile diarrhœa, the incidence among breast-fed children being extremely low. The disease frequently spreads with alarming rapidity. Dietetic upsets as the result of concomitant disease or improper feedings predispose to infection. Children with pink disease are particularly vulnerable.

Pathology and Clinical Pathology.

The morbid anatomy and histopathology do not differ from those occurring in adults and already described. However, subacute and chronic forms of the disease are rare in children and as a rule only the acute stages are seen, since either the infection is rapidly fatal in the first few days or cure results.

Since the disease occurs so often in a hyperacute form, biochemical changes, reflected by the clinical picture, are much more striking than in adults. As the result of vomiting, which is more common and more persistent than

in adult dysentery, and the choleraic type of diarrhœa, there is a considerable loss of fluids and chlorides. More importantly, base, chiefly in the form of sodium, is lost in large amounts in the stools, and in conjunction with the development of inanition, which leads to imperfect fat metabolism, severe acidosis develops in many cases.

In the isolation of organisms, rectal swabs are of considerable advantage. The swab is examined microscopically for an exudate and plated out in the usual way.

Clinical Features.

The onset is usually sudden, with vomiting, violent purging, abdominal colic and rapidly developing prostration. The stools show at first undigested curds, become greenish and finally are small and fluid. In the severe attacks blood and mucus appear, but there is usually fæcal material, an important point in the clinical differentiation from intussusception. Within a short time the child becomes drowsy, with a high temperature (in hyperacute cases this may be subnormal). The skin rapidly loses its normal elasticity, the face appears old and shrunken, the eyes are sunken, and the fontanelle depressed. With attacks of colic the child cries and draws up the legs. As the disease progresses and acidosis appears, futher dehydration is evident, subcutaneous fat is diminished and air hunger is apparent. The urine is scanty, of high specific gravity, and strongly acid. In the more severe cases cyanosis appears. Persisting hiccup is of grave prognostic significance.

Death may occur within the first few days. Recovery is accompanied by a reduction in the number of stools and lessening of the blood and mucus present. The skin regains its normal tone, vomiting ceases and the urinary output rises.

The clinical picture generally is a caricature of the disease occurring in adults. Signs and symptoms are more intense, and with the rapid march of symptoms death may come swiftly. Recovery may be rapid.

Diagnosis.

It is obviously of considerable importance to determine the ætiological factors. Diarrhœa due to *otitis media* or other infections may closely follow the course described above. It is therefore necessary to search carefully for an upper respiratory tract infection or inflammation of the middle ear and mastoid. There is no doubt that diarrhœa with colitis does occur as the result of parenteral infection, but, in the writer's opinion, many of the more severe cases are the result of a bowel infection and treatment should never be entirely guided by the finding of a septic focus.

Dietetic upsets may also cause severe diarrhœa, but again, not infrequently, an enteral infection coexists.

Intussusception may be mistaken, but with this condition the stools rapidly change to blood and mucus in which, however, the inflammatory characteristics of bacillary dysentery do not appear. A tumour is felt early in the majority of cases.

Diagnosis requires the detailed bacteriological investigations already given, in conjunction with the clinical signs.

Treatment.

The prophylactic value of breast feeding is of the greatest importance.

Management of the case is concerned with appropriate dieting, fluid replacement, correction of the deranged acid-base equilibrium, treatment of coexisting septic foci, and specific treatment directed at the dysenteric organisms.

Once a diagnosis is made, the sooner sulphaguanidine is started, the better. In the more acute cases vomiting will interfere with its ingestion. In this case an intramuscular injection of one of the soluble sulphonamides should be given and repeated at eight hourly intervals. The infection will in this way be controlled and toxicity diminished; oral administration of the sulphonamide of choice then is usually possible.

The diet for infants follows, in the main, the principles already laid down for adults. At first water only is given in small frequent feeds. Glucose 10% and half-normal saline solution is a useful adjuvant. After a day or so skimmed dry milk is given, again as small feeds and at frequent intervals. The next addition is sugar (saccharose). Protein hydrolysate forms the next addition; and finally the proportion of fat is increased in the feeds. The clinical condition and daily inspection of the stools will be the guide for these changes in diet. Retrogressive steps in the diet may sometimes be necessary. With any severe degree of dehydration intravenous administration of fluids is imperative. The administration of fluids subcutaneously into the fontanelle and into the peritoneum has been largely superseded by intravenous administration and should seldom be used now. Even the smallest babies, by the use of a needle[1] designed by Collins,[14] can be given fluids continuously by the intravenous route. The infusion should be given slowly. The fluid of choice is glucose 10% in normal saline solution. If acidosis is marked, four molar sodium lactate should be injected into the tubing. It is seldom necessary to give fluids intravenously for more than a day or two.

In milder cases of dehydration, subcutaneous injections of half-normal saline solution are effective.

Amœbic Dysentery.

Epidemiology and Ætiology.

Distribution.

Amœbic infection follows in some cases the ingestion of *Entamœba histolytica* cysts. These develop into trophozoites which invade the mucosa of the large bowel, producing characteristic pathological changes. By absorption into the portal veins the amœbæ may infect the liver and other organs. Our main concern here is the dysentery which results from bowel infection, although dysentery as such is not the most common result of amœbic activity.

The disease is known to occur all over the world, but is more frequent in the tropics, probably because of (*a*) a climate favourable for cyst longevity, (*b*) the low standard of living, and (*c*) the primitive excretory habits of the inhabitants.

In the history of warfare it has never occupied the dominant position of the dysentery group, although some outbreaks have been serious. The incidence seldom exceeds 10% and usually is only 2% to 5% of all overt dysenteries in the army. In the Australian Military Forces during 1943-1944, 784 cases (including relapses) were reported. In the same period 7,500 cases of bacillary dysentery were notified.

The infections are not related to any particular season.

Epidemic waves are not common. Exceptional epidemics, for example that which occurred in Chicago in 1933, may be of considerable magnitude. This outbreak produced at least 1,400 cases with 52 deaths. However, the disease is much less amenable to permanent cure than the bacillary group; treatment is longer and more arduous; and the disease leads to considerable chronic ill health in its victims. Amœbiasis is often unsuspected and even when considered in a diagnosis amœbæ may be difficult to detect.

Method of Spread.

The disease is spread by the usual means of infection in intestinal diseases: indirectly by flies (and possibly rodents and cockroaches) and directly by the ingestion of water or food infected by fæces containing *Entamœba histolytica* cysts. While the trophozoites are delicate organisms, with little resistance to an inimical environment, the cysts, enclosed in a capsule, resist chlorination beyond a concentration compatible with drinking, and can survive up to a month so long as conditions are favourable. Drying rapidly leads to their death. After ingestion the cysts change to trophozoites which invade the large bowel mucosa and give rise to dysentery.

Carriers.

For some reason not well understood, symptomless amœbiasis, even in civilian populations, is common. These

[1] This needle has been successfully used to give a transfusion to a baby weighing 0·68 kilogram (one and a half pounds).

amœbæ are indistinguishable morphologically from those which cause dysentery. Surveys in different parts of the world have shown that from 2% to 15%—sometimes up to 40%—of the population harbour *Entamœba histolytica* cysts in their stools with no apparent deterioration of health in the majority. These healthy carriers—or contact carriers—probably play a part in the epidemiology of the disease. Major H. O. Lancaster (personal communication) has made a detailed study of the incidence of amœbæ among Australian troops, completed over a period of five months. The results are shown in Table V.

TABLE V.

Place of Exposure.	Number Examined.	*Entamœba histolytica* Cysts.	
		Number.	Percentage.
Australia	155	5	3·2
Middle East	95	10	10·5
New Guinea	569	21	3·7
Middle East and New Guinea	185	19	10·0
Total	1,004	55	5·5

This valuable survey shows that the carrier state is equally common in Australia and New Guinea. It is not easy with these data, showing the same carrier rates in the two countries, to account for the rarity of amœbic dysentery in Australia and its occurrence in New Guinea, where about 3·5% of all dysenteries were amœbic. It may be that bacillary dysentery, occurring as an epidemic, allows the amœbic carrier state in some cases to develop into an overt attack due to damage to the intestinal mucosa, which allows a portal of entry for the amœbæ. The size of the dose, general resistance of the patient and pathogenicity of the amœba may all have their effect.

It has been questioned whether the cysts in all these people are pathogenic; and indeed it is possible that different strains of *Entamœba histolytica* exist, varying in their virulence and pathogenicity. Knowing how readily other intestinal infections will gain momentum and produce widespread epidemics with only a few carriers at large, one wonders why, if all cyst carriers are dangerous, this disease should not also cause many cases in conditions favourable for an epidemic. Possibly only some strains will cause the disease. Granting, alternatively, that all strains of *Entamœba histolytica* are pathogenic, the explanation for its sporadic occurrence may lie in some of the known facts of experimental infection of volunteers. Walker and Sellards,[15] by feeding cultures of amœbæ to 21 healthy men, showed that not all the men become infected and that a minority developed dysentery (four of the eighteen "parasitized"). It may well be that host resistance varies greatly, and this may account partly for the anomaly in incidence between amœbic and bacillary dysentery.

However, Faust[16] in 1941 reported on early autopsies of 202 people dying suddenly from accidents. Evidence of *Entamœba histolytica* infection was found in 13 (6·44%) and amœbic lesions, although superficial and minimal, were demonstrated in five of these, suggesting that resistance is only relative.

It may be that convalescent carriers, who, as opposed to contact carriers, constitute a very small percentage of the population, form the main group which maintains the disease. There can be no doubt of the pathogenicity in these cases.

Parasitology.

A number of different amœbæ are found in human excreta. Of these, only *Entamœba histolytica* is known with certainty to be pathogenic.

As it occurs in the fæces and tissues, the trophozoite of this amœba is usually 20 to 30 thousandths of a millimetre in diameter. The organism is actively motile and indeed the speed of its movement is one of the main distinguishing points. Its high refractility distinguishes it from macrophages and other tissue cells. The amœba is phagocytic and, unlike all non-pathogenic species, readily engulfs red blood corpuscles. The endoplasm is finely granular and contains the nucleus, which, however, cannot always be seen. Vacuoles are seldom observed. The nucleus is very fine—"mapping-pen" drawn—with a circular ring of chromatin along which are set fine chromatin dots. The karyosome is not prominent. The ectoplasm is highly refractile, and the manner of pseudopod formation characteristic. Large blunt processes are put out; after a short delay, and when the pseudopod is quite large, the endoplasm flows into it. The trophozoites are usually found in active dysentery, and always in the tissues, when ulceration has occurred.

Entamœba coli, with which *Entamœba histolytica* is most likely to be confused, can be distinguished by the following characteristics. Usually it is larger than *Entamœba histolytica* (20 to 50 thousandths of a millimetre), but small strains do occur. Red blood corpuscles are never found engulfed. In fresh specimens it is far more sluggish than *Entamœba histolytica*. The nucleus is built on the same plan as in *Entamœba histolytica*, but the chromatin dots are much coarser. The ectoplasm is not so prominent as with *Entamœba histolytica*, and in movement the pseudopods are not so well marked, the endoplasm appearing to flow into a bulge almost as soon as it is formed. Vacuoles are common.

The cysts of *Entamœba histolytica* formed from the trophozoites are about 10 to 15 thousandths of a millimetre in diameter and have the type of nucleus described above. Each mature cyst contains from one to four nuclei. In the cytoplasm are usually found chromidial bars. These are fairly large and cigar-shaped, and have rounded ends. They rarely are found in stools with blood and mucus. They never appear in the tissues.

The cysts of *Entamœba coli*, which form from the trophozoites, may closely resemble those of *Entamœba histolytica*. However, the nucleus still preserves the coarse chromatin beading of the nuclear membrane; there may be one to eight nuclei, usually four to eight; and the chromatoid bars, which are not nearly so prominent, are "splintered" and thread-like.

Pathology and Clinical Pathology.

Morbid Anatomy.

The Large Bowel.—As with bacillary dysentery, the pathological changes in the large bowel can best be studied in the living state through the sigmoidoscope.

Amœbic cysts on their passage through the alimentary canal change into trophozoites. These invade the large bowel (rarely the distal end of the ileum as well). The main incidence, as seen in fatal cases (Clark,[17] 1925), is in the caecum and ascending colon or the whole colon. The initial attack is through the crypts of Lieberkühn, although, where the resistance of the host is high or the virulence of the amœbæ low, the lesion may be restricted to microscopic breaks in the epithelium. These minimal lesions, it has been pointed out, may occur in the symptomless contact carrier state. Should the processes continue, ulcers are formed, which extend deeply, involving usually the submucosa, but in others going on to destroy all tissues down to the peritoneum. At first the ulceration occurs by virtue of the lytic properties of the amœbæ. Later, secondary infection continues the destructive process, in combination with the trophozoites.

The ulcers, at first shallow, become flask-shaped in the submucosa and finally deep, necrotic and with overhanging edges. Earlier star-shaped or circular, the more advanced ulcers are irregular in outline, and, if extensive, may merge into each other. They are surrounded by hyperæmic or hæmorrhagic zones. There may be only a single ulcer, or there may be many. Areas of submucosal hæmorrhage only may be seen, or these may appear in between actual ulcers. In some cases a gangrenous process of the large areas of bowel occurs with inevitably fatal results.

Characteristically, the intervening mucosa in moderate cases of ulceration is normal to inspection through the

sigmoidoscope. However, it has been pointed out that such an appearance may be seen with subacute and chronic bacillary dysentery.

The ulcers by extension may cause perforation with abscess or fistula formation. Gross hæmorrhage may occur as the result of ulceration. In rare cases a large, firm, granulating mass, called an amœboma, may result. This has been mistaken for a carcinoma. With healing considerable fibrosis takes place if the condition has continued for a long time. Where treatment is given fairly early and is adequate, the ulceration completely heals, leaving behind no visible trace.

The Liver.—Amœbæ may be carried by the portal veins and gain lodgment in the liver. Two types of amœbic infections of the liver are seen clinically—amœbic hepatitis and amœbic abscess. Abscess formation results from a gradual extension of the morbid processes which begin as amœbic hepatitis.

With the latter condition the resistance of the liver tissues is overcome, lytic substances derived from the amœbæ break down the cells and minute areas of necrosis result. Should these extend, or in the absence of treatment, coalescence will ultimately produce an abscess. Usually they are single, but several may occur. Rarely multilocular abscesses are found. The abscess is found about ten times as commonly in the right lobe as in the left, and hypertrophy occurs in the unaffected portions of the liver. Secondary infection may occur at any time, or the abscess spontaneously may become encapsulated with absorption, obliteration and finally scar formation.

Early, free trophozoites are found, but in long-standing abscesses, the amœbæ tend to die out and are seldom found. They can nearly always be found in the walls of the cavity. The contents at first are like anchovy sauce, owing to the presence of autolysed liver tissue. Later, with secondary infection, thick pus, stained brown or green, is formed. Up to a gallon or more may be drained from a single abscess. In neglected cases perforation may occur with fatal peritonitis.

Other Organs.—Next to the liver, the lungs are the most commonly affected organs. The infection may be by the blood stream via the portal system or, more commonly, by direct upward extension through the diaphragm from the liver. The lung abscess has characteristics similar to those described in the liver. Obviously the right lower lobe will most commonly be affected. The sputum is streaked with material like anchovy sauce. No authentic case of amœbæ being found in the sputum has been recorded (Manson-Bahr[18]). Death may ensue, but with appropriate treatment or even spontaneously resolution with scarring may occur.

Rarely other organs are invaded by amœbæ. The brain may be involved, usually in association with amœbiasis of the liver. The fifth case to be reported (Halpert and Ashley[19]) occurred in a man whose amœbic dysentery had been present for four years. *Entamœba histolytica* was demonstrated in an occipital abscess. Other organs, including the spleen, testes and the urinary tract, are rarely involved.

Histopathology.

The two main effects of amœbic infection on the tissues are lysis due to enzymes excreted by the organism and thrombosis of small blood vessels. This can be seen in microscopic sections where single trophozoites appear (always in rounded form in stained sections) surrounded by zones of lysed tissue, or in large numbers in the coalesced lesion. Section may show amœbæ lying in the capillaries, and by this means they gain access to the portal system, which must be common. The tissues respond to infection, not by a polymorphonuclear response, which is always evidence of secondary bacterial infection, but with histiocytes.

When ulceration occurs, section shows the earliest change to consist of invasion of the surface epithelium to produce minute lesions. Should that extend, a flask-shaped ulcer is produced which by extension may be converted to a deep ulcer with overhanging edges. At the boundaries of the ulcer there is considerable œdema, and in this area most of the trophozoites are found.

Cysts do not invade the bowel wall. They are found superficially on the mucosa embedded in the mucus, and therefore cannot be influenced by emetine given by injection.

Clinical Pathology.

It requires emphasis that, in a search of fæces or bowel scrapings for amœbæ, the fresher the specimen, the better the chances are of recognition and the fewer the chances of mistaking non-pathogenic amœbæ for *Entamœba histolytica.*

The macroscopic appearance of the fæces in the acute stage may differ not at all from the fluid stool with blood-streaked mucus seen in bacillary dysentery.

The same technique for detection, described in regard to bacillary dysentery, is used. If mucus is present, this is selected for microscopy, for therein lies the best chance of finding amœbæ. The exudate may be very similar to a typical bacillary one, but usually differs in the paucity of polymorphonuclear cells. There are often numerous red cells and degenerated tissue cells. Charcot-Leyden crystals, while by no means diagnostic, appear fairly commonly in amœbic stools and are a useful pointer to possible infection.

To demonstrate amœbæ a warm stage to the microscope is not necessary so long as the stool is absolutely fresh. In the acute stages usually trophozoites only are seen, and these are recognized by the points enumerated earlier. Rapid movement, engulfed red cells, delicate nuclei and blunt pseudopods are the main differentiating features from *Entamœba coli.* It is important not to mistake feebly motile macrophages containing red cells for amœbæ.

It has been pointed out that, even when amœbæ have been demonstrated, it is still wise to culture the stool, since a bacillary infection may coexist.

The search for amœbæ in a stool having the characteristics mentioned should be repeated many times if amœbiasis is suspected on clinical grounds. In the acute stages, however, this difficulty is not pronounced.

In cases of chronic dysentery, and in cyst carriers, the search may need to be lengthy. If lesions are visible with the sigmoidoscope, and no amœbæ are found in the stools, the ulcers should be gently scraped with a curette and the material examined. Even in the absence of visible lesions scraping of the mucosa will sometimes reveal amœbæ when stool examinations have given negative results. Administration of a saline purgative before the stool is examined may aid in the detection of the organisms. "Provocative" emetine injections are useless.

In cases of amœbic hepatitis or abscess, about half the patients have amœbæ in their stools (Manson-Bahr[18]).

Symptomatology.

Amœbic infection of the large bowel is protean in its manifestations. The infection may be initiated by acute diarrhœa, similar to severe bacillary dysentery, from which the patient may die in a short time if untreated; or the original infection may give rise to so slight an attack of diarrhœa that medical advice is not sought until the disease becomes chronic. Moreover, infection undoubtedly can take place without the patient's being aware of a bowel upset at any time. All grades of severity between these two extremes may be encountered.

Acute Amœbic Dysentery.

The incubation period varies considerably. Walker and Sellards,[15] in their experiments with volunteers, found in four cases incubation periods ranging from 20 to 95 days. However, the Chicago epidemic showed that this could be as short as seven days. The author had a patient admitted whose incubation period was ten days or less.

He was a nursing orderly who had been working in the dysentery ward, where there were patients suffering from amœbiasis for ten days before the acute onset of symptoms. Amœbic dysentery was not endemic in the locality, the patients in the ward having come from forward areas to which he had not been. Three days before his admission to

hospital diarrhœa had started. He passed five loose stools the first day, four the second and three the third. No blood or slime was noticed. Soon after the onset, abdominal colic and tenesmus started, he felt nauseated and headache developed and he had a mild rigor. On examination there was no abdominal tenderness. The first specimen examined was of normal appearance and contained no exudate or amœbæ on microscopy. Six days after onset his stools were liquid and a typical bacillary exudate was found. No pathogens were isolated on culture. *Entamœba histolytica* trophozoites were swarming. Next day, sigmoidoscopy showed slight thickening and granularity of the mucosa with moderate generalized hyperæmia. There were no erosions or ulcers. The patient was afebrile throughout.

The symptoms in this case might easily be mistaken for bacillary dysentery, especially as tenesmus is uncommon in amœbic dysentery. The onset is usually sudden in the acute cases, and although five to ten is a usual number of bowel actions daily, the author has seen patients with 15 to 20 bowel actions daily.

Fever is uncommon in amœbic dysentery unless some complication such as amœbic hepatitis is present.

Abdominal pain is often vague and usually occurs in the lower part of the abdomen. Colic may occur. Tenesmus is uncommon, but in the author's experience occurs more often than is generally stated. There is usually some abdominal tenderness, mainly over the sigmoid colon.

The fæces are liquid and may or may not contain blood and mucus. No reliance can be placed on the gross appearance in the making of a diagnosis from other forms of dysentery, although large amounts of blood in the fæces are more common with amœbic than with bacillary dysentery.

Sigmoidoscopy in the early acute case may reveal little abnormal. The bowel may show hyperæmia only. Later, ulceration develops. There may be only one or two; there may be very many ulcers.

Chronic Amœbic Dysentery.

Chronicity is a marked feature of amœbic infections. In many cases the amœbæ appear to live in the host's bowel with little or no constitutional disturbance. In others, vague malaise and symptoms referable to the abdomen—including pain, colic, dyspepsia, and intermittent attacks of mild diarrhœa—signal a smouldering infection. A minority of patients suffer severely from a chronic infection with active ulceration of the large bowel, recurrent attacks of dysentery and constitutional symptoms of considerable severity.

At any time, in any of these cases, amœbic hepatitis, amœbic abscess of the liver or other complications may supervene.

Accordingly we may classify chronic amœbiasis into four main clinical categories: (i) contact carrier state (symptomless), (ii) chronic or recurrent dysentery, (iii) amœbic hepatitis and amœbic abscess, (iv) amœbiasis of lung and brain.

Contact Carrier State.—Contact carriers have already been dealt with. They are discovered by stool surveys.

Chronic or Recurrent Dysentery.—The severity of cases in the category of chronic or recurrent dysentery will vary from a mild recurrent diarrhœa which causes little or no constitutional symptoms, to a severe intermittent or continuous diarrhœa which leads to a condition of cachexia. The symptoms may be confined to diarrhœa and some abdominal pain; but in many cases there is often epigastric discomfort or pain, accompanied by a capricious type of dyspepsia.

In cases of moderate severity diarrhœa is complained of and often blood and mucus are noticed in the unformed stool. The patient seldom feels completely well. The appetite is poor, epigastric discomfort or pain is common after a meal, and lower abdominal pains may occur when diarrhœa is present. Weight is lost and loss may be extreme. Acute exacerbations of dysentery may occur.

A small number of patients suffer from severe continuous dysentery, with gross ulceration of the bowel, lasting many months and ending in death. They may be resistant to anti-amœbic treatment and the possibility of gross secondary infection causing this intractable condition must be considered. Treatment will be modified in these cases.

In a small group of eight consecutive cases of recurrent amœbiasis of slight to moderate severity, the author has found that jolting in vehicles usually induced pain or abdominal discomfort, sometimes diarrhœa and vomiting. The sensation is usually described as a heavy dragging feeling in the right hypochondrium and epigastrium. Often, too, the heavy discomfort in the right side is noticeably worse after a long day's march or heavy labour. Table VI gives some features of these ten patients.

TABLE VI.

The Effects of Jolting in Amœbiasis on Bowel Actions and Abdominal Pain.

Case Number.	Stools.	Diarrhœa.	Accentuated Pain.
1	*Entamœba histolytica* cysts ..	Nil	+
2	*Entamœba histolytica* cysts ..	Nil	+
3	*Entamœba histolytica* cysts ..	Nil	+
4	*Entamœba histolytica* cysts ..	Nil	+
5	*Entamœba histolytica* trophozoites	+	+
6	*Entamœba histolytica* trophozoites	Nil	+
7	*Entamœba histolytica* trophozoites	Nil	+
8	*Entamœba histolytica* trophozoites	+	+

These symptoms also occur to some extent with functional diarrhœa and functional dyspepsia, but not with the same regularity. Diagnosis in some cases has been helped by eliciting this story. In only one case were the symptoms volunteered. It may be that in all these cases amœbic hepatitis was beginning, but there was no other evidence of this, except for slight tenderness in the epigastrium and right hypochondrium in some.

In this group occur a proportion of cases with dyspeptic symptoms which may be difficult to diagnose. When chronic dyspepsia is complained of by people who have been exposed to amœbic infection, consideration should always be given to the possibility of amœbiasis, especially if a history of recurring bowel upsets is given, no matter how mild. Some patients may give a clear history of acute dysentery, and failure of response to sulphaguanidine may give a lead to the diagnosis. However, the absence of any history of acute dysentery by no means excludes the possibility of amœbic infection. In the army the story given in these cases is often as follows:

The soldier had been in the Middle East and New Guinea. In common with nearly everyone with foreign service, he has had attacks of diarrhœa, and perhaps one or more of frank dysentery. He has also had several attacks of malaria. For several years he has had recurrent attacks of mild diarrhœa, but no blood or slime is seen. He frequently complains of epigastric discomfort, sometimes of pain coming on half to one hour after meals. His appetite is poor, he has not regained all the weight he lost in New Guinea, and he has little energy for, and less interest in, his job. On examination there is some diffuse epigastric tenderness. The liver may be enlarged for two fingers' breadth and is slightly tender. There is vague diffuse lower abdominal tenderness and the sigmoid may be palpated. In addition there are home worries and some stigmata of a neurosis.

These are indeed difficult cases. Repeated stool examination and sigmoidoscopy give negative results. Investigation by X rays reveals no abnormality. There is no anæmia or leucocytosis.

In some of this group of men *Entamœba histolytica* cysts are found. There is little doubt that in many cases the patient is merely a contact carrier with an additional neurosis. However, the organisms, if found, cannot be ignored. They may be the cause of his symptoms or may flare up into overt dysentery. Moreover, only by specific treatment will it be shown whether the patient is essentially neurotic or whether he is suffering from amœbic infection.

Amœbic Hepatitis and Amœbic Abscess.—When a section of the bowel wall, infected by *Entamœba histolytica*, is examined by the microscope, frequently amœbæ are seen lying free in the lumen of small blood vessels. It seems

probable that, in every case of amœbiasis in which there is any degree of mucosal invasion, the liver receives a constant bombardment of amœbæ carried by these vessels to the portal system. Apparently, though, the liver in the great majority of cases can deal with these. In fact, spontaneous cure may result after even massive involvement and abscess formation. Probably the abdominal discomfort, mild dyspepsia, flatulence and anorexia, complained of in so many cases of mild amœbiasis, have their origin in minimal liver involvement. However, in a percentage of cases, varying in different parts of the world, and depending on the efficiency of early treatment, liver involvement occurs which dominates the clinical picture.

The resulting illness varies from the very mild to the very severe. In mild cases the patients complain of vague dyspepsia or inconstant attacks of upper abdominal discomfort and pain. The more severe case follows in the main a recognizable pattern of symptoms and course, but undoubtedly the condition may be obscure and baffling.

The onset is insidious. The patient usually gives a history of recurrent attacks of dysentery. This is not always so; some patients are unaware of any previous bowel upset. At first there is epigastric and right hypochondriac discomfort, sometimes with exacerbations and the occurrence of quite severe pain. The patient often describes the sensation as an aching, nagging discomfort. Some complain of pain on jolting or on jumping from a height. The pain is sometimes worse on deep breathing. The general health declines, appetite is lost, and the weight falls, sometimes to a remarkable degree. There may be short bouts of fever accompanied by rigors. The patient looks sick, and often has a muddy complexion. On examination little may be found in the early case, except enlargement and tenderness of the liver. In cases of active colitis, when attacks of dysentery recur, there is usually tenderness in the iliac fossæ. It is not common to find basal pulmonary signs at this stage. The significance of the enlarged and tender liver is sometimes difficult to assess in a sample of the population exposed to malaria. Examination of the blood will in some show a moderate polymorphonuclear leucocytosis. This becomes more pronounced as the disease advances and with abscess formation is invariably found. Fluoroscopy in early cases of amœbic hepatitis seldom shows any restriction of diaphragmatic excursion. The stools, after repeated examination, may yield no amœbæ and the sigmoidoscopic appearance of the bowel may appear normal.

In the most severe case, abdominal pain may increase rapidly over a few days. Usually epigastric, constant, but subject to exacerbations, it is characteristically very severe, and requires morphine for its control. Fever mounts and usually remits or intermits, sometimes with rigors, the pulse rate rises, and pain may be complained of in the shoulder of the affected side through irritation of the diaphragm. Sometimes the pain is made worse by deep breathing and changes in posture. Usually the tenderness, which is acute over the liver, precludes any assessment of liver size. Crepitations or râles are frequently heard at the lung base of the affected side, and fluoroscopy will commonly reveal some abnormality of diaphragmatic movement.

It is at this stage, or even in the milder earlier cases mentioned, that a correct diagnosis is so important. Treatment is very satisfactory and will prevent the disease from progressing to abscess formation.

In some cases, however, massive destruction of the liver may proceed insidiously to abscess formation, and the first indication is rupture. In some a subphrenic abscess occurs. In others, when the diaphragm is penetrated, a pulmonary abscess forms.

This may communicate with a bronchus, forming a hepatico-bronchial fistula. Large amounts of pus, often like anchovy sauce, are coughed up. Hæmoptysis may or may not occur. Less commonly, perforation occurs into the abdominal cavity, giving rise to a general peritonitis. Most patients, though, will pass through the stages described and, if untreated, develop an abscess which can usually be diagnosed. With the onset of secondary infection the condition becomes more serious. The temperature is more hectic, pain more severe, and leucocytosis of 30,000 to 40,000 cells per cubic millimetre with 80% to 90% polymorphonuclear cells occurs.

With abscess formation fullness of the epigastrium may be discovered on inspection. The liver is found to be greatly enlarged both by palpation of its lower edge and by percussion of its upper limits. Tenderness is usually very acute. In addition, fluoroscopy will show often one or more of the following signs (Fairley[20]): (*a*) diminished movement or "splinting" of the right or, more rarely, the left cupola of the diaphragm; (*b*) elevation of the right or left cupola of the diaphragm; (*c*) abnormal upward projection or "humping" of the cupola of the diaphragm, indicating that an abscess is pointing toward the lung; (*d*) abnormal signs at base of right or left lung, indicative of collapse, consolidation, effusion or abscess; (*e*) an area of increased density in the liver itself, occasionally evident in central liver abscess.

Pain may be complained of on springing the ribs and in the more advanced cases the finding of a localized tender spot is of help in diagnosis. Through this region exploratory puncture should be directed and aspiration attempted. For this a wide-bore needle, not longer than three and three-quarter inches to avoid injury to the inferior *vena cava*, is used. Local anæsthesia is satisfactory. One attempt to locate the abscess frequently fails, and several punctures in a number of directions may be necessary. A description of the pus has been given.

Finally, if no pus is detected, or if it is thought that the condition is amœbic hepatitis without abscess formation, a therapeutic trial with emetine injections is indicated. The results are dramatic. Pain and fever rapidly subside, and after three to six days of treatment the patient is convalescent.

Amœbiasis of Lung and Brain.—It was pointed out that involvement of organs other than the large bowel and liver are uncommon. Lung involvement may be (*a*) primary due to a blood-borne infection; or (*b*) secondary from upward extension through the diaphragm.

With primary infections, the gradual onset of pain in the chest, cough, fever and physical signs of consolidation may be the presenting syndrome. Hæmoptysis may or may not occur. Occasionally an acute onset simulates pneumonia.

In secondary infections, following an illness such as is described above, rupture through the diaphragm will lead to a pulmonary abscess. Extension of this into a bronchus results in the expectoration of large amounts of amœbic pus. Basal adhesions usually prevent the occurrence of empyema.

When the brain is involved symptoms of a cerebral tumour arise and although a correct anatomical diagnosis may be made, it is obviously difficult to determine the pathology.

Diagnosis and Differential Diagnosis.

In a discussion of the diagnosis, a number of other conditions with which amœbiasis may be confused have been mentioned. Further points will be added here and other diseases considered. So long as the condition is borne in mind and, in cases of doubt, every use made of the diagnostic procedures mentioned, mistakes are not likely to be made.

In the acute stage the difficulties are fewer. Recognition of the case as one of dysentery should always lead to a correct differentiation from bacillary dysentery, with which it is most likely to be confused.

In the chronic stage the problem is more difficult. The differential diagnosis may be considered under the headings already given. All these symptoms may occur in the same patient at different times, but usually one or other syndrome dominates the clinical picture.

The Chronic Carrier State.

Contact carriers are symptomless and have been discussed in a previous section.

Chronic and Recurrent Diarrhœa.

Chronic Bacillary Dysentery.—The diagnostic features of chronic bacillary dysentery have been described in a previous section.

Ulcerative Colitis.—It may be difficult to rule out ulcerative colitis. The points in diagnosis have been considered in connexion with chronic bacillary dysentery. It should be emphasized that if any doubt exists emetine should be tried.

Carcinoma of the Colon.—Growths of the right side of the large bowel commonly are of the medullary type, which ulcerate and give rise to attacks of diarrhœa alternating with constipation. The stools may closely resemble those of dysentery. These symptoms, occurring in the older age groups and in the absence of a demonstrable cause in the stools or on sigmoidoscopic examination, always demand a barium enema and X-ray examination.

Tuberculosis of the Large Bowel.—Tuberculosis of the large bowel may be part of generalized glandular tuberculosis, as seen in children and native races, or secondary to open pulmonary lesions when large numbers of tubercle bacilli are swallowed. The symptoms resulting are those of chronic diarrhœa with an exudate in the stool. Sigmoidoscopy may show ulceration. Repeated examination of the stools for tubercle bacilli will make clear the diagnosis in most cases.

Diverticulitis.—The vague lower abdominal symptoms occurring with amœbiasis are frequently present in cases of diverticulitis. The stools may contain blood, pus and mucus. If a mass is felt in the left iliac fossa and amœbæ are absent from the stool, a barium enema and an X-ray examination will be necessary to exclude this condition.

Polyposis Intestini.—The symptoms in polyposis disease may imitate closely those of chronic amœbic dysentery. However, there is usually a family history and a barium enema will show the tumours.

Amœbic Hepatitis and Abscess.

If the pain occurs in the lower part of the abdomen, recurrent appendicitis may be considered, and also diverticulitis. Upper abdominal pain, such as occurs with amœbic hepatitis and amœbic liver abscess, may give rise to diagnostic difficulties which tax the most astute physician.

Acute and Chronic Cholecystitis.—From the description given of the disease it can be readily seen how closely acute or chronic cholecystitis may be simulated. In some cases, and in the absence of amœbæ in the stools, the author knows of no certain differentiating points short of a cholecystogram. A therapeutic trial with emetine may be necessary.

Cholangitis.—Jaundice, rarely present in amœbic liver infection, occurs in cholangitis, which usually follows inflammation of some abdominal viscus. Pain, tenderness, fever, rigors, pulmonary basal signs and leucocytosis are common to both.

Subacute Pancreatitis.—An acute attack of pancreatitis with a dramatically sudden onset can hardly be mistaken. With milder recurrent attacks there may be confusion. Estimations of the urinary diastase are of value.

Hydatid Disease of the Liver.—Hydatid disease of the liver needs consideration, especially in Australians, since it may closely mimic an amœbic abscess. It must be remembered that large sections of the community had been previously exposed to hydatid disease before the war and to amœbic infection since it started.

The symptoms commonly follow the same pattern, with epigastric fullness on inspection, pain, tenderness, fever, leucocytosis and pulmonary signs and symptoms. Eosinophilia is much commoner in hydatid disease than in amœbic infection. By the time a hydatid cyst is infected, the Casoni test and the complement fixation test may give negative results. If an abscess is diagnosed, then exploratory aspiration is called for. If there is doubt about the presence of an abscess, the effects of emetine should be tried.

"P.U.O."—The early case of amœbic hepatitis is frequently diagnosed as malaria. Blood films show no parasites and, as in all cases of doubt, the effects of antimalarial treatment should be tried. The presence of a leucocytosis will often make the diagnosis of malaria unlikely. Typhus, typhoid fever, undulant fever, renal tract infection and tuberculosis and septicæmia may all need to be considered.

In one patient with typhoid fever in whom no rash appeared, and whose symptoms were mainly upper abdominal pain and tenderness, the author has misdiagnosed amœbiasis and given emetine. Failure of response to treatment and the finding of a positive blood culture led to the right diagnosis.

An epidemic of pleurodynia (Bornholm disease), in the author's experience, at first caused considerable anxiety before it was recognized.

The first patient admitted to hospital complained for several days of malaise, with pain in the right hypochondrium, made worse with breathing and on lying on the right side. On examination there was fever, with tenderness over the liver, and at the right lung base, fine crepitations. The syndrome lasted for a week. All investigations, including blood examination, fluoroscopy, stool examinations, and cholecystogram, gave negative findings. A further attack in a fortnight added to the suspicion of an amœbic hepatitis.

Other patients with the syndrome were admitted to hospital at this time and it was then recognized that this case was the first of an epidemic which affected about 100 troops. The "crepitations" heard were almost certainly caused by a fine friction rub which occurred in 10% of the cases.

Amœbiasis of the Lung.

The finding of basal pulmonary signs has led to the exhibition of sulphonamides and the death of patients from an unrecognized amœbic liver abscess (Fairley[20]). Attention to the points already mentioned should prevent this mistake. Lung abscess, with or without the occurrence of hæmoptysis, may be the presenting sign. The response to emetine is diagnostic. Lung abscesses from other causes (hæmtogenous, hydatid disease, bronchiectasis, foreign bodies) need to be differentiated.

Treatment, Management and Prognosis.

In emetine we have a specific drug for the treatment of amœbiasis. With large numbers of cases occurring among service personnel there have been published a series of disillusioned reports about the efficacy of the drug. Lamb and Royston[21] (1945) state that, after combined treatment with emetine ("Auremetine" given orally), "Carbarsone" and "Quinoxyl" enemata, 91% of 29 patients suffered a relapse within five months of treatment. Their patients had nearly all been treated before, some of them many times, and, as they point out, do not therefore represent the average case.

Such unhappy results need not always be anticipated if the treatment initially is thorough and the patients are treated early in the disease. Only a minority—about 10%—will relapse under these conditions.

There can be no single standard form of treatment for use in all cases. But from a therapeutic point of view we may consider them under the four main headings previously mentioned.

The appropriate courses of treatment will therefore be as set out under the four following subheadings.

Primary Acute Amœbic Dysentery.

Days 1 to 10:

Emetine hydrochloride (one grain in one millilitre of water) for ten injections. (Patient strictly in bed.)

Days 11 to 32:

No treatment.

Days 33 to 42:

Emetine-bismuth-iodide ("E.B.I.") orally each evening at 2030 hours, preceded one hour by one grain of phenobarbital.

Dosage, first day ..... grain 1
Dosage, second day ..... grains 2
Dosage, third day ..... grains 3
Dosage, fourth to tenth day .... grains 3 daily

Sodium bicarbonate (2% solution) bowel washout followed by "Yatren" (six to eight ounces in 2·5% solution) retention enema: to be given each morning and retained until 1700 hours with suitable posturing.

Days 43 to 49:

"Carbarsone" 0·25 gramme twice daily for seven days.

Days 50 to 55:

Six specimens of fæces examined for *Entamœba histolytica*.

There are two points about this course of treatment which need emphasis. Failure to carry out all the details may well lead to failure in response and the occurrence of a relapse.

The "E.B.I." capsules, especially in the tropics, where they become hard, may pass unchanged through the gut (Manson-Bahr[20]). They should therefore be pricked with a pin to aid their solution. Needless to say, it is necessary to check any vomiting which occurs, as the capsules frequently give rise to considerable nausea.

The retention enema is given very slowly, to avoid any tendency for it to be returned, with the patient in the left lateral position. The foot of the bed is then raised on blocks nine inches high. For the first half-hour the patient should lie on the left side to assist in the distribution of the solution. After that the patient lies supine for another half-hour. Thereafter he may choose his own position in bed.

Emetine is a toxic drug and throughout the course the patient is confined to bed. However, in the average case occurring in healthy young adults some latitude is allowed and the patient may be permitted to use a bedside commode.

The drug is slowly excreted and for this reason a full course of emetine should not be followed by "E.B.I." until three weeks have elapsed. This combined course undoubtedly decreases the incidence of relapse (Manson-Bahr[22]). In many cases the relapses are the result of inadequate treatment in the first instance.

Relapsing or Chronic Amœbic Dysentery.

There is little to be gained by giving numerous emetine hydrochloride injections in cases of chronic amœbiasis when the patient is passing cysts. It is possible that repeated courses of emetine will give rise to "emetine-fast" strains. Moreover, emetine, by injection, has no effect on cysts. The course for these should start at the thirty-third day of the above scheme of dosage.

With active chronic or relapsing amœbic dysentery, in which trophozoites are found and ulceration is present, the course may with advantage start with emetine hydrochloride by injection for three or four days until diarrhœa is controlled. "E.B.I." and "Yatren" may then follow straight on.

It has recently been suggested (Hargreaves[23]) that the treatment of the small group of patients with severe, intractable, amœbic dysentery which fails to respond to emetine should include penicillin and sulphasuxidine in an endeavour to control secondary infection of the bowel. Hargreaves's results are so encouraging that this supplementary treatment should be given widespread trials for such cases.

Cyst Carriers (Symptomless).

The course should start at the thirty-third day on the above scheme for cyst carriers without symptoms.

Amœbiasis with Liver or Lung Involvement or Both.

In amœbiasis with liver or lung involvement or both the full course should be given. Even if no bowel lesion has been demonstrated or if no amœbæ have been found one cannot rule out the possibility of a still smouldering infection of the colon in areas inaccessible to vision.

Should the infection have proceeded to abscess formation, drug therapy usually is not sufficient, especially if secondary infection occurs. Except in the case of patients who are desperately ill, when immediate aspiration is imperative, it is wise to give emetine by injection for several days before aspiration of the abscess. The chance of hæmorrhage is thereby reduced (Manson-Bahr[18]).

For its performance the patient should be given a preliminary injection of a quarter of a grain of morphine and one one-hundredth of a grain of hyoscine. The choice of site for aspiration may be influenced by the radiological appearances or by local signs, such as bulging, œdema of the skin or tenderness confined to one area. However, if there are no indications, the mid-axillary line on the right side at the eighth intercostal space will be the initial site of entry.

The skin and chest wall are infiltrated down to the pleura with 2% "Novocain" solution. The abscess having been located with a stout, wide-bore needle (not longer than three and three-quarter inches), the aspirated contents are examined. Should material like anchovy sauce be drawn, having no smell and containing no visible pus, the aspiration is completed by a Potain's aspirator. The pus should be examined for bacteria by cultural methods. The advantage of injecting emetine when the contents are evacuated is doubtful.

If, on preliminary aspiration, the contents contain obvious pus and are foul smelling, aspiration is completed, but before withdrawal, 50,000 units of penicillin are injected into the cavity. The material should be cultured and, if a penicillin-sensitive organism is recovered, penicillin should be given parenterally as well as emetine.

If the symptoms and signs do not subside, and the leucocyte count remains high, aspiration should again be tried in about a week. More pus may be found in the aspirated cavity or, if this proves dry, further punctures may find a second abscess.

In cases of abscess of the lower surface of the liver, abscess of the left lobe, or where the pus is too thick for aspiration, an open operation may be necessary. The mortality is much higher than with aspiration, and operation should be avoided whenever possible; with modern treatment it is very rarely indicated.

Criteria of Cure.

Clinical Criteria.

Treatment with "E.B.I." and "Yatren" usually results in the patient losing half a stone or even a stone in weight. It is important, therefore, to see that all this weight, and usually more, is regained before his discharge.

There should be no complaint of abdominal pain or tenderness. The appetite and sense of well-being should be normal.

Fæces.

Daily inspection of a stool is important, to observe the result of treatment and to ensure that the "E.B.I." is diffused through the stool, which then appears very dark. The stools should be formed, normal and without blood or mucus. For one week after the last dose of "Carbarsone" six stools should be examined microscopically for any exudate or cysts.

Sigmoidoscopy.

Should the patient not be fully cured, there will usually be some indication of failure from clinical findings or fæces examination. However, on occasions there is no evidence other than that afforded by examination with the sigmoidoscope, which should always be done.

It must be remembered that amœbiasis is a chronic disease and that latency may still persist when all these criteria have been fulfilled. "Cured" amœbiasis is a relative term and only time can show if the phrase is absolute. For this reason a check on the clinical condition, fæces and sigmoidoscopic appearances is worth while at three-monthly intervals for six months and at six-monthly intervals for the next year. Hepatitis may be diagnosed early thereby, abscess formation prevented, and precautions against active chronicity taken.

Iodamœbiasis.

Iodamœba butschlii is possibly pathogenic. In a Japanese prisoner of war who died in Australia, Dr. E. H. Derrick demonstrated at autopsy lesions in the stomach, colon, lungs and brain. The colon showed ulceration similar to that found with *Entamœba histolytica*. The liver was not

involved. In the lesions amœbæ were found which Dr. C. M. Wenyon states are identical morphologically with *Iodamœba butschlii* (Brigadier N. Hamilton Fairley, personal communication). Unfortunately, no report on the stools was available.

These findings have stimulated a search for patients with iodamœbæ (trophozoites or cysts) in their stools who may be suffering from the effects of infection.

Iodamœba butschlii trophozoites are sluggishly motile and contain a small nucleus with most of the chromatin concentrated in the karyosome. Red cells are never contained by them. The cysts have usually a small single nucleus—sometimes two—with the same features as in the trophozoite, and a compact mass of glycogen. There are no chromatoid bodies. The organism is found in a percentage of normal people, varying from less than 1% up to 14% (Wenyon and O'Connor[130]).

Major H. O. Lancaster (personal communication), in a stool survey of 849 soldiers who had been in the Middle East, New Guinea, or both, found that 19 (2·4%) harboured cysts of *Iodamœba butschlii*. None were found in 155 men who had never left Australia.

The author has seen four persons infested with *Iodamœba butschlii* in whom there were abdominal symptoms. The main clinical and laboratory features are given in Table VII.

None had diarrhœa at the time of admission to hospital. Sigmoidoscopy and fluoroscopy of the diaphragm in each case gave normal findings. The total white and differential white cell counts were essentially normal.

A medical officer (Case IV and Table VII) had his stools examined ten months before his admission to hospital—four months after an attack of frank dysentery which had not responded to sulphaguanidine—because of recurring diarrhœa and abdominal discomfort. *Iodamœba butschlii* cysts were found.

These symptoms had continued and he was admitted to hospital. Iodamœbæ cysts were again found. "E.B.I." was given and the stools became normal. In the following two months he had two mild attacks of diarrhœa, but felt well. One year later he was quite well and diarrhœa had not recurred.

It has not been possible to learn the fate of the other three men. The early clinical response to treatment by two of them was not very satisfactory. It is well known that *Iodamœba butschlii* is highly susceptible to emetine, as these cases bear out. With the satisfactory elimination of the organism and complete relief of the symptoms, one still cannot be certain that the syndrome is due to *Iodamœba butschlii*. A latent infection with *Entamœba histolytica*, which is relieved, could account for any improvement that occurs.

It is possible that *Iodamœba butschlii*, like *Entamœba histolytica*, varies profoundly in its relation with a host. Some patients may become symptomless carriers, but the infected Japanese prisoner of war suggests that in others a severe infection results.

Further observations are required to settle the question of pathogenicity. In the meantime patients with a history of recurrent diarrhœa and abdominal symptoms suggestive of amœbiasis should receive a course of "E.B.I." if *Iodamœba butschlii* is found in the stools.

BALANTIDIUM COLI: ISOSPORA HOMINIS: MALARIA: KALA-AZAR.

Balantidium Coli.

Ætiology.—Although it appears to have a world-wide distribution, infection with *Bacillus coli* is rare. The organism is a common parasite in the bowel of pigs and monkeys (Manson-Bahr[125]). The disease occurs mainly among those whose work brings them into close association with pigs.

This ciliate is about 50 to 80 thousandths of a millimetre in diameter and 50 to 70 thousandths of a millimetre broad. It can be seen readily with the low power of a microscope. At the anterior extremity is the periostome—a depression which leads into the mouth. The ectoplasm is covered with cilia by which the organism moves in a rotary and forward direction. There is a large nucleus more or less central and two contractile vacuoles. Multiplication is by transverse fission or conjugation. The organism may form a resistant encysted stage.

Pathology.—The organism invades the bowel wall in a similar manner to *Entamœba histolytica*, by virtue of a lytic enzyme, producing hyperæmia, hæmorrhages, and finally ulcers with undermined edges.

Symptomatology.—The disease is notable for its chronicity. Clinically the symptoms are similar to amœbic dysentery, especially in its chronic form. There is diarrhœa, continuous or in attacks (the stools often containing blood and mucus); abdominal colic, tenderness and constitutional disturbances occur, in some cases leading to anæmia and cachexia. The disease may last many years.

Overt disease possibly is not always present when the organism is found. The author has seen a New Guinea boy admitted to hospital with Flexner dysentery in whom trophozoites of *Balantidium coli* were present. Sigmoidoscopy two weeks later, after sulphaguanidine, showed the bowel wall to be normal and the patient was clinically cured. No *Balantidium coli* could then be found.

Diagnosis.—Diagnosis is made by stool examination when the organism will be found or scrapings from ulceration seen at the sigmoidoscopy. The discharge of organisms may be intermittent, so that several stools should be examined.

Treatment.—The numerous drugs advocated for its treatment indicate that balantidiosis is difficult to cure. Emetine, silver nitrate, protargol, oil of chenopodium, "Yatren", methylene blue, and "Stovarsol" have all been used with varying success.

Isospora Hominis.

Ætiology.—Intestinal coccidiosis caused by *Isospora hominis* is a very rare disease. It has a world-wide dis-

TABLE VII.
Iodamœbiasis.

Case Number.	Past History of Dysentery.	Exposure.	Symptoms.	Signs.	Fæces.	Days Before Stools Cleared with Emetine-bismuth-iodide.
1	In New Guinea and Middle East.	Middle East and New Guinea.	Indigestion and pain under right costal margin for six months.	Tender over liver.	Iodamœbæ cysts on six examinations.	3
2	Nil.	New Guinea.	Indigestion for twelve months.	Nothing abnormal detected.	Iodamœbæ cysts on six examinations. Vegetative forms once.	3
3	Middle East and New Guinea.	Middle East and New Guinea.	Intermittent mild diarrhœa for three years and pain under right costal margin.	Tender over liver.	Iodamœbæ cysts on seven examinations. Vegetative forms twice.	2
4	New Guinea.	New Guinea.	Intermittent mild diarrhœa for one year.	Liver one finger's breadth and tender.	Iodamœbæ cysts on six examinations.	2

tribution. Up to 1935 only 209 cases had been reported (Magath[25]). The organism is a parasite of the intestinal epithelium, and in the cells the life cycle occurs. This, as with the malarial parasite, consists of shizogony and sporogony.

Infection in Man.—The disease causes diarrhœa, abdominal discomfort and minor constitutional symptoms. Oocysts are found in the fæces. Apparently no exudate is found in the stools (Manson-Bahr[18]). Treatment is satisfactory with bismuth salicylate.

Malaria.

In some severe cases of falciparum malaria, gastro-intestinal symptoms are prominent. Dysenteric features are those which concern us here.

In New Guinea in 1942, when quinine was the suppressant used for malaria, the author had in his care approximately 800 patients with malaria, of whom 60% were suffering from falciparum infections. Ineffective suppression allowed many severe infections to develop, and intravenous administration of quinine was not infrequently required. In spite of this there were no cases of dysentery occurring in malaria in which the symptoms could unequivocally be ascribed to the plasmodium.

In patients with malaria who developed symptoms of dysentery, it was usual to find bacillary dysentery coincident. Although there is no doubt that a clinical condition of dysentery does result from the parasites sporulating in the vessels of the large bowel, one cannot help feeling that the occurrence is rare.

In countries where falciparum malaria occurs, mistakes are unlikely to be made. Every patient admitted to hospital, for whatever disease, under these conditions should have at least one thick film examined. Severe infections, sufficient to cause dysenteric symptoms, will almost invariably be diagnosed correctly in this way.

If diarrhœa or clinical dysentery is present as well, the stools should be examined microscopically and cultured. Sulphaguanidine would then be given concurrently with antimalarial treatment.

The perils of ascribing diarrhœa in cases of malaria to the plasmodium are well illustrated by the Shiga infection mentioned in a previous section.

Kala-Azar.

In advanced infections with *Leishmania donovani* ulceration of the large and small bowel may occur. The clinical picture of a chronic febrile illness, with anæmia, leucopenia, pigmentation, splenomegaly and hepatomegaly is obviously at variance with that of chronic bacillary or amœbic infections.

Examination of the blood, bone marrow and splenic puncture will usually reveal the parasites.

Helminthic Dysenteries.

Schistosomiasis.

Ætiology and Epidemiology.

Three species of these trematodes are parasitic to man: *Schistosoma hæmatobium*, *Schistosoma mansoni* and *Schistosoma japonicum*. Only the last two give rise to dysenteric symptoms and will mainly concern us here.

It is not proposed to deal other than very briefly with the ætiological agents, of which full descriptions are given in standard textbooks. Suffice it to say that the trematodes are unisexual, the female being enclosed in a groove on the ventral surface of the male. The adult worms live in the veins. *Schistosoma mansoni* and *Schistosoma japonicum* live mainly in in the portal and mesenteric veins, while *Schistosoma hæmatobium* is found in the pelvic (bladder, uterus, prostate) and the lower mesenteric veins. The female leaves the male and enters the small venules. Eggs are laid there and lie in a manner resembling a "string of sausages" (Manson-Bahr and Fairley[20]). Thence they escape into the bladder or intestine.

Eggs may be found in the many organs—lung, liver, spleen and brain. The distribution varies with the species (Manson-Bahr[18]). *Schistosoma hæmatobium* is found in pelvic viscera, lungs, brain and spleen; *Schistosoma mansoni* is found in the colon, small bowel and liver; *Schistosoma japonicum* is found in the intestine, mesenteric and retroperitoneal lymph glands, liver, lung and brain.

Eggs ejected in the patient's urine or fæces, on coming into contact with water, hatch into miracidia, which for their further development are dependent on species of fresh water snails, and these vary with the different schistosomes. In them the cercariæ develop and are discharged. By contact with infected water the skin is penetrated by cercariæ which gain access to the lymphatic or blood vessels.

Pathology and Clinical Pathology.

The essential pathological changes are the same with each of the three species of schistosomes. The differences in pathology and symptomatology are expressions of the varying choice of habitat. The earliest change is a phlebitis and results from toxins liberated by the organisms. Hatching of the eggs is followed by the development of nodules consisting of small round cells, eosinophile cells, monocytes and giant cells surrounding the ova (Fairley[27]). The eggs do not all escape into the lumen of the viscera attacked. Some nodules become fibrosed and eventually calcified.

With the passage of the eggs through the epithelium excess mucus is formed and hyperæmia and scattered hæmorrhages become evident. As the result of this dysenteric symptoms may arise. Sigmoidoscopy will in the early stages reveal this hyperæmia and excess mucus. The small nodules may also be seen. Later these may develop into pedunculated adenomatous growths. Should these slough off, ulcers will be produced. Malignant changes may occur in the tumours.

Symptomatology.

In the early stages of invasion the clinical manifestations may be separated into four stages (Fairley[27]):

1. *Dermatitis.* Local itchiness may develop as the result of cercarial invasion. A rash may occur, lasting a day or two.

2. *Toxic or Anaphylactoid Features.* Toxic and anaphylactoid features appear about two months after infection and consist of generalized urticaria and transient œdema associated with high eosinophilia. Fever may last for a number of weeks and be associated with rigors. Deposition of the eggs in the lungs will give rise to respiratory symptoms and, with heavy infections, to a bronchopneumonic condition. There are epigastric pain and vomiting, and the liver becomes enlarged and tender. The spleen may be enlarged.

3. *Dysenteric Features.* Diarrhœa may occur from the third month and may be very slight or severe, with the passage of blood and mucus in the stools and marked tenesmus. Constitutional symptoms become marked. Loss of weight and anæmia appear.

4. *Cirrhosis.* Cirrhosis of the liver with ascites, hepatomegaly, splenomegaly and cachexia mark the terminal stages.

From the second or third month on other complications may be evident. Chronic pulmonary fibrosis and involvement of the brain or spinal cord may occur. Vesical invasion, with hæmaturia and chronic cystitis, is characteristic of *Schistosoma hæmatobium*, which, however, does not give rise to dysenteric symptoms.

Diagnosis.

A careful history is important, since the disease occurs only in certain well-defined localities.

In the early stages the methods available are Fairley's complement fixation test and intradermal sensitivity test. In the case of patients exposed to the disease, who show a clinical picture mentioned under the toxic and anaphylac-

toid symptoms, these tests should always be performed. High eosinophilia with a total leucocytosis is suggestive.

The result of the complement fixation test becomes positive from the second week of infection and in nearly all cases is positive in the first three months. The result of the dermal sensitivity test is not so often positive.

If the diagnosis has not been made in the early stages by these means, the onset of diarrhœa in about the third month will lead to stool examination and sigmoidoscopy. From the mucus of the stool, or from a swab, ova can be demonstrated microscopically. This is done by examining the mucus under a coverslip with the low power. If some of this mucus is placed in water at about 35° C., miracidia will escape and can be seen with a hand lens.

Concentration methods may be necessary for demonstrating ova when they are scanty.

Treatment.

For treatment of schistosomiasis, antimony, which kills the adult worms, is specific. With *Schistosoma mansoni* and *Schistosoma hæmatobia*, organic compounds, such as "Fouadin", are said to be satisfactory. For *Schistosoma japonicum*, however, sodium antimony tartrate (tartar emetic) is the most satisfactory drug.

The solution must be made up on the day of injection. Injections are given every second day, a start being made with half a grain in ten millilitres of distilled water, and the dose being increased half a grain each injection until the maximum of two grains is reached. A total course is 30 grains.

Some side-effects of the drug may occur. There may be a cough soon after injection and nausea or even vomiting may occur.

For polyposed bowel masses or for tumours around the anus, surgical treatment in addition to drugs may be necessary.

DYSENTERY IN THE ARMY.

Incidence.

Although the Australian Military Forces have not suffered from dysenteric diseases so severely as in the last war and the infections have not been so chronic, nevertheless this group has ranked second only to malaria as a cause of wastage. In Egypt and Libya the disease at times reached alarming proportions, while in the earlier New Guinea campaigns the existence of at least one force was threatened by dysentery. Only by the prompt exhibition of sulphaguanidine in all regimental aid posts was the epidemic halted. In a later campaign, with a force of about 100,000 men, there were 3,157 cases of diarrhœal disease, and the vast majority were cases of bacillary dysentery. Sulphaguanidine was freely available, and this incidence was a great reduction on earlier rates.

One interesting feature of dysentery is the occurrence of some immunity to the whole bacillary group from infection by one type. Thus it is found that more than one attack of proven bacillary dysentery is uncommon in the same person. Recent experimental evidence on animals suggests that there is overlapping immunity with the various types. The "salting" process, so often observed in troops, may therefore have a real basis.

When many cases are occurring, a proportion show the clinical features of bacillary dysentery, the stool contains a typical exudate, but no organisms are isolated. One has always assumed that these were clinical attacks. However, experience of diarrhœal diseases among troops living in good conditions of hygiene in Australia has made one question the truth of this assumption. When only sporadic cases were occurring in North Queensland among 70,000 troops in 1944, dysentery organisms were rarely isolated. Clinically, the disease was mild and indistinguishable from bacillary dysentery. In spite of an intensive search for organisms—dysentery and salmonella—they were not isolated. No doubt these cases occur in epidemic conditions and constitute a small submerged proportion. It is possible that these cases are in fact due to bacillary dysentery, but in the author's opinion this is unlikely. Their ætiology remains unproven.

Control.

It has been pointed out that, for the checking of dysentery epidemics, there are a number of lines of attack. Briefly, we have these methods: (*a*) carrier control, (*b*) control by drugs, (*c*) sanitation.

An epidemic is susceptible to attack by all these methods, which should be used simultaneously.

Some points on the first two methods will be mentioned here.

Carrier Control.

A list of the main types of dysentery carriers has been given, and it has been pointed out that the incompletely recovered were the most dangerous and the most numerous. In an epidemic, once the integrity of the water supply has been proved and fly control instituted, should the disease not cease almost immediately (within a few days), then every means available should be used to detect carriers. Kitchens, messes and ration stores are the places on which attention should be concentrated. All food handlers are examined individually, a history is taken, and a rectal swab is collected and immediately plated on desoxycholate-citrate medium. By this method exact bacteriological information can be obtained and all carriers isolated and treated. Careful bacteriological and clinical control is necessary before they return to their units.

Such methods, however, are not always possible in the field. A more crude method, which in the author's experience has yielded good results, was first described to him by Major J. D. Hicks. From every man of the units in which dysentery is occurring, a specimen of fæces is examined macroscopically. This can easily be arranged by issuing each man with an empty two-pound jam tin with the lid removed, into which he passes his fæces. On this tin is a label which bears the soldier's number and name. When collected, these are examined by the medical officer. Reasons have been given for the view that in an epidemic of dysentery the main sources of danger are those who pass frequent, abnormal stools. Therefore, in sorting out the stools passed, those which are formed and normal are discounted. Close scrutiny of all men who pass abnormal stools will pick out the dangerous carriers.

An example of the application of this technique occurred in a small epidemic at Milne Bay, in New Guinea, investigated by the author. The hygiene and sanitation generally were irreproachable and a search was therefore instituted for carriers. Additional evidence for the infection being due to a food handler was provided by two companies (A and B), who marched out during the epidemic, after which they had only one case. All food handlers had previously been questioned about their bowel movements; all had denied diarrhœa. Stools were collected from every man as above (62 men). The following results were observed: formed normal, 35; soft formed, 20; fluid with or without mucus, 7.

These last seven were immediately evacuated. Six of them had developed diarrhœa in the previous twenty-four hours. It was found that the remaining man was the sergeant cook who had previously denied any diarrhœa. Faced with the evidence of his own excreta, he then admitted that for six months, following an attack of dysentery, he had constant diarrhœa with abdominal discomfort and loss of weight.

After this man was evacuated the epidemic immediately ceased. Table VIII shows the number of cases.

No laboratory was in the area at this time, so that bacteriological information is not available.

Control by Drugs.

There is no doubt that, for the mass control of a serious epidemic, sulphaguanidine will achieve remarkable results. It may happen, as on the Kokoda Trail in New Guinea, that an epidemic gets out of hand and that circumstances make the observance of even elementary hygienic principles extremely difficult. In these conditions every case of diarrhœa should be regarded as one of bacillary dysentery and treatment with the drug should be instituted at

TABLE VIII.

Weekly Periods.	Companies.				Totals.
	Headquarters.	A.	B.	Motor Transport.	
January 26 to February 1	2	3	2	0	7
February 2 to February 8	2	2	0	0	4
February 9 to February 15	1	3	2	0	6
February 16 to February 22	1	1	0	1	3
February 23 to March 1 ..	4	0	0	1	5
March 2 to March 8 ..	12	1	0	8	21
March 9 to March 15 ..	1	0	0	0	1
March 16 to March 22 ..	0	0	0	0	0
March 23 to March 29 ..	0	0	0	0	0
Total	23	10	4	10	47

February 22: A and B companies marched out.
March 7: Stool inspection.
March 9: Last case occurred.

the earliest possible moment. The cooperation of all ranks must be obtained for this. By this method the disease is cut short or actually aborted, carriers are diminished, and fæces exposed to flies become non-infective. There is no doubt that in this campaign a critical situation was saved by its use.

ACKNOWLEDGEMENTS.

This account of the dysenteries is mainly a personal one based on my experiences of the diseases as they occurred in the Australian Army in Australia, the Middle East and New Guinea. Like the great majority of army medical officers, confronted for the first time by this group of diseases as the result of the war, I started with an abysmal ignorance of the subject. What I have since learnt and here presented owes a great deal to my senior officers and my colleagues.

Particularly I am indebted to Brigadier N. Hamilton Fairley, whose experience, wisdom and encouragement have been invaluable. To Colonel E. V. Keogh, Lieutenant-Colonel E. Britten Jones and Lieutenant-Colonel F. H. Hone I owe my first acquaintance with the subject and have been guided and encouraged by their interest. I acknowledge also my indebtedness to Major A. A. Ferris, Major H. O. Lancaster and Captain Ian Jones for their generosity in pathological guidance and clinical assistance.

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APPENDIX.

Sigmoidoscopy.

Stress has been laid, throughout this account of the dysenteries, on the importance of sigmoidoscopy for diagnosis and the final survey of a patient. The technique for using the sigmoidoscope is not difficult, nor does it demand the surgical ritual so often given to its use. The essential points in the procedure for diagnosis and follow-up in cases of dysentery are as follows.

1. *Preparation of the Patient.*—No preparation is required in the majority of cases. Early morning is the best time for those passing normal stools, since in most cases the rectum and sigmoid are then empty. When diarrhœa is present, the time of election is immediately following a bowel action. In some cases a simple bowel washout with normal saline solution at body temperature is all that is required. Paraffin should never be given beforehand.

2. *Posture.*—Without question the knee-left shoulder position is best, with the patient acutely flexed. The patient should be examined on a table four feet high. He rests on his knees and on the left shoulder, with the left arm adducted under his body.

3. *The Instrument.*—It is an unnecessary refinement to heat the sigmoidoscope to body temperature. The important point in its passage is the liberal use of "Vaseline", which should be smeared thickly on its tip and about the distal six inches of the tube.

4. *Passage of the Instrument.*—The instrument is gently pushed through the anal orifice until the sphincter is overcome, when it readily passes. After about ten centimetres are inserted, the obturator is withdrawn and the light and eye-piece are fitted. Then under direct vision the instrument is passed, usually for a distance of about 25 centimetres. It is rarely necessary to inflate the colon with bellows for this.

5. *Collecting Specimens.*—A long swab is passed through the instrument. It is made of stiff wire with a sterile piece of cotton-wool twisted on its end. If ulcers are present, these are gently swabbed, and the swab is plated on culture medium. Reddened areas and erosions may be gently rubbed to see if bleeding occurs. In a search for amœbæ, a small blunt curette mounted on a long wire is useful. With this, scrapings are gently taken from any ulceration and examined microscopically as soon as possible. If ulceration is not present, an attempt should be made to collect some mucoid secretion from the bowel wall, an area of hyperæmia being chosen. Amœbæ may be found in this way when both swabs and stool examination reveal none.

Reviews.

INHALATION THERAPY.

THOUGH, as a monograph, it may appear rather long to the busy reader, "Inhalation Therapy" contains much material of interest alike to general practitioner and consultant.[1] Ideas on the subject of inhalation are too often limited to the use of oxygen or carbon dioxide, or both, in a variety of conditions, and to the use of steam with or without an adjuvant drug. This volume will broaden knowledge of the possibilities of this form of therapy and will rationalize the procedure in more conditions. The book is so set out that an introduction to each subject is followed by the pathological physiology of the condition to be treated and the actual inhalation treatment of the condition itself. Nor is inhalation therapy stressed in importance at the expense of other and more accepted methods, but in each case it is placed in what appears to be its true perspective in relation to other forms of treatment. Adequate information is given to those inexperienced in therapy of this type, in regard to the type of gas and drug combinations to be used and the apparatus available for their administration. Sixty-five pages towards the end of the book are devoted entirely to a description of apparatus, to the mechanics of its operation and to its care and maintenance.

An introductory chapter on the historical and physiological aspects will stimulate interest in the rest of the volume, and modern problems of aviation medicine are described in the second chapter. Special sections of particular interest deal with the use of inhalation therapy in bronchial asthma and in pulmonary emphysema. These sections give the reader valuable information; in them, and indeed throughout the book, graphs and figures give a readily recognizable physiological basis for the principles of therapy set out. Other chapters deal with inhalations in pneumonia, pulmonary œdema and congestive heart failure, coronary thrombosis and sclerosis, and shock. War medicine receives consideration in chapters on the treatment of gas poisoning with lung irritants and blast injuries of the lung, and an entire chapter is devoted to the description of varieties of respirators. Throughout there is an extensive bibliography to aid further reference, and the whole presents an extremely comprehensive survey in a form which makes ready access to its sections possible.

THE TREATMENT OF ACCESSIBLE CANCER WITH X RAYS.

ANOTHER new book from the British school of radiotherapy has come to hand. This volume by D. Waldron Smithers is entitled "The X-Ray Treatment of Accessible Cancer".[2] The author has used exclusively low voltage X-ray therapy apparatus, from which relatively low depth doses are obtained. Thus only surface or near surface tumours of proven radiosensitivity offer suitable material. The preliminary chapters deal with the principles of treatment, and the types of apparatus available are discussed.

An excellent chapter on relevant radiation biology is included in which the author presents a method of histopathological examination of a series of sections taken during progress of treatment of surface neoplasms. Here the proportion of potentially active to impotent cells is estimated. These examinations help in determining optimum dose increments, over-all time and total dosage, but unfortunately no general conclusions are put forward, nor is guidance given in regard to treatment factors for the varieties of cases met with in clinical practice. Here a few well-chosen photomicrographs would have been of great help to the reader.

The author passes to consideration of neoplasms of the skin, mouth and external genitalia; tumours arising in these sites and their subdivisions are discussed. In every instance a brief but satisfactory clinical description is presented, excellent photographs of illustrative cases before and after treatment are provided, and full tabulations of results are given. But problems of treatment complications and technical details are not produced for similar effective consideration.

The results of treatment of skin, lip and mouth carcinomata show that radiation is the most useful form of treatment in these sites, but nevertheless there is a certain proportion of failures. This fact makes effective initial treatment essential, and stern warning is given about inadequate radiation of small rodent ulcers.

Glands of the neck should not be removed for prophylactic reasons following squamous-cell carcinoma of the lip if the primary growth is small and specialized; but dissection is indicated if the primary growth is large, or anaplastic, or if operable glands are present.

The author has used superficial X-ray therapy for treatment of secondary glands of the neck, but this appears to be a practice of very dubious value in view of the small fields and low depth doses available. Here the indications and contraindications for such treatment should be more fully considered, and one cannot accept the dictum that it is necessary to treat individual glands instead of gland fields. The reader can only conclude that other forms of radiation are more suitable for the work in hand.

The work concludes with a chapter on lymph drainage, and here are presented several statements at variance with accepted teaching.

The author is to be commended upon an excellent piece of work, the keynote of which is the application of accurate physical method to a branch of clinical radiational practice, and he tells a story of successful effort. The physical details of various arrangements are carefully depicted in numerous isodose charts, the publication of which is most welcome. Nevertheless, the radiotherapist is left somewhat disappointed at sins of omission, as much more technical detail would have been appreciated. A chapter on mould room accessories would have enhanced the text. The illustrations are very numerous and of high quality, especially the colour plates. The printing and binding are the work of craftsmen.

THE PROBLEMS OF FAMILY LIFE.

"THE PROBLEMS OF FAMILY LIFE", by Agatha H. Bowley, Ph.D., is a small volume of 98 pages which could be read with considerable profit by all who have the welfare of children at heart—parents, teachers, social workers, nurses and doctors.[1] The book is based primarily on Dr. Bowley's experience of the difficulties of parents and children as senior psychologist to the Leicester Education Committee.

The positive elements necessary for happy family life are discussed in conjunction with the relationships between mothers and fathers, between parents and children, and between brothers and sisters. Fundamental problems concerning jealousy, discipline, freedom and punishment, and the need for affection and security are adequately dealt with. Practical suggestions are given for good social, intellectual and emotional outlets for the various age groups.

Dr. Bowley stresses the necessity for parent education in any scheme of reconstruction, fully realizing the difficulties that can arise when parents are unstable, over-ambitious or harassed by material wants. Her remarks about the impossibility of teachers assisting children with individual personality problems when they have classes of fifty or more are unfortunately only too true of Australian conditions here as well as of those overseas.

Many writers have dealt with the treatment of delinquent children, and Dr. Bowley's analysis of her various case studies is full of interest; but in spite of recent increases, delinquents are still a small percentage of children, whereas most children at some time present behaviour problems and habit disorders which if unwisely handled can lead to much unhappiness for all concerned. It is in the discussion of these everyday problems which beset all who deal with children that Dr. Bowley's understanding of causes and of positive common-sense treatment is particularly valuable.

Suitable play material for various ages is listed as an appendix, and there is a series of photographs showing different stages of development.

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[1] "Principles and Practices of Inhalation Therapy", by Alvan L. Barach, M.D.; 1945. Oxford: Blackwell Scientific Publications, Limited. 9" × 6", pp. 334, with 59 illustrations. Price: 25s. net.

[2] "The X-Ray Treatment of Accessible Cancer", by D. Waldron Smithers, M.D., D.M.R.; 1946. London: Edward Arnold and Company. 10¾" × 7½", pp. 156, with many illustrations, some coloured. Price: 40s.

[1] "The Problems of Family Life: An Environmental Study", by Agatha H. Bowley, Ph.D.; 1946. Edinburgh: E. and S. Livingstone, Limited. 7¼" × 5", pp. 114, with twenty photographs. Price: 5s. net, postage 5d.

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PARALYSIS AGITANS AND ITS SURGICAL RELIEF.

WHEN Parkinson described the distressing condition known as *paralysis agitans*, he referred to "involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace; the senses and intellects being uninjured". Kinnier Wilson, who quotes this description[1] observes that Parkinson makes no reference to rigidity and that the word in fact does not appear in his essay; by implication, however, it underlies the posture in flexion to which he refers. Wilson points out that the first inroads of the malady are so slight and nearly imperceptible that sometimes the patient can hardly recollect the precise period of onset. He describes a trifling general slowness and stiffness of movement and a tendency to maintain position for an unusual length of time. To this becomes added a proneness to trembling in one of the hands or arms or, rarely, in some other part. It is common for early manifestations to be one-sided. Once they have appeared, stiffness and tremor as a rule gradually increase and spread. Eventually the classical triad of symptoms, tremor, rigidity and flexion attitude, are present, and on these other symptoms depend. In the matter of treatment Wilson points out that it is worse than useless to administer any kind of nerve tonic to "whip up" a patient's decaying cells, but that rather should some readily assimilable pabulum be sought in the hope of supplying from without what the cell itself cannot obtain from within. Experience shows only too well that this hope is vain, and Wilson admits as much. He states that all other treatment is purely symptomatic or even more empirical. There the matter has remained and the clinician has taxed his ingenuity to the full to provide some measure of relief to the unfortunate sufferer. Recently some attempts have been made to give surgical relief to the patient. Though some work has been done, the reports are few and some of these have appeared in journals that are not readily available. It is unfortunate that not so many months ago prominence was given in a magazine that is widely read by the general public to a series of operations performed by one investigator. The matter was described in such a way that non-medical persons were led to believe that the operative procedure had become established and that good results might be expected to follow its performance. Clinicians in the several States of the Commonwealth have, because of this glamorized account, which should never have been published, been asked by patients or their relatives whether "the operation" could not be performed for them. In these circumstances it has seemed advisable to describe the present status of surgery in the disease known as *paralysis agitans*.

In regard to the pathology of *paralysis agitans*, Wilson points out that in 1911 he drew attention to the analogies between it and progressive lenticular degeneration, which is a striatal disease *par excellence*, and declared that its pathology must be sought in the same general region. Lewy in the following year described chronic cell atrophy and glial overgrowth in putamen and *globus pallidus* with reduction of fibres in the *ansa lenticularis*; he held these changes resembled lesions of senility. Later he observed that most of the atrophic cells showed senile fibrillar change; he also laid stress on the great cellular loss of the *globus pallidus*, declaring that cells of the *substantia nigra* were regularly involved. Small foci of rarefaction in the *corpus striatum*, optic thalamus and external and internal capsules, with disappearance of cells from the thalamus and *corpus Luysii* and fibre poverty in the *globus pallidus*, were reported by Auer and McCough. Wilson concludes his discussion on the pathology of the condition with the general statement that the primary basis of the cardinal symptoms is a degenerative process attacking the *corpus striatum* and the nuclei connected with it. With this knowledge of the pathology it is possible to understand the occurrence of rigidity and tremor. The reciprocal relationship between the basal ganglia and the cortex is disturbed and the cortex assumes a dominating role. This influence of the cortex has to be interrupted if such a condition as tremor is to be relieved. T. J. Putnam, who has been prominent in work on this subject, has stated[1] that P. C. Bucy reported the first successful operation for the relief of alternating tremor in 1938. In this case severe tremor of the left hand followed a head injury and relief was afforded by removal of the contralateral arm area in the motor cortex down to the depths of the central sulcus. The result was a cortical monoplegia which the patient found that he preferred to his tremor. Putnam refers to a large series of cortical ablations that have been performed by R. M. Klemme on account of tremor. The only account of Klemme's work that is available is the reprint of an article published by him in *Modern Medicine*, of March, 1945. It consists largely of a series of line drawing showing the surgical procedure adopted by him. We read that the procedure was devised by him, that he has used it in the treatment of 200 patients suffering from Parkinson's disease, and that by its means 78% of them

[1] S. A. Kinnier Wilson: "Neurology", page 790.

[1] "Encephalitis: A Clinical Study", by Josephine B. Neal; 1942. London: H. K. Lewis and Company Limited.

have been successfully rehabilitated. A footnote to the title of the article states that it has been reproduced from a film prepared by Klemme in conjunction with the Medical Film Guild of New York City. This may account for the absence of any detail to support the statement of 78% recovery. The operation, Klemme states, is based on the concept of a continuous premotor cortico-spinal nerve bundle. Resection of the premotor cortex prevents stimuli from passing down the bundle and spreading to other tracts. Klemme points out that the common-path cells of the spinal cord are responsible for muscle movement. The common-path cell depends on the internuncial cells (which are under direct control of the voluntary motor centres, but subject to modification by sensory influences) for 70% of its contacts, on the premotor cortex for 10% and on all others for 20%. When the body is at rest and the overwhelming motor influence is absent, normal muscle tone is maintained by cooperation between the basal ganglia and the premotor impulses. In Parkinson's disease the basal ganglia are the site of pathological change; premotor activity is therefore unrestrained and tremor results. (This is the dominance of the cortex that has already been mentioned.) According to this concept, removal of the premotor influence by surgical excision restores normal functions, or should do so. Putnam (*loco citato*) states that Klemme has had a mortality of 17·5%. He also states that in some cases Klemme's operations were bilateral. Putnam goes on to remark that the amount of cortex removed may be reduced to a minimum by exposure of the brain under local anæsthesia and by the injection of "Novocain" in one region after another until the tremor is controlled. The crucial area, he states, is the summit of the precentral convolution (Area 6), close to the mid-line. The more restricted the extirpation, the milder will be the disability. Putnam holds that there are difficulties in affecting a tremor of the leg by this method, but no information is available to show what claims Klemme makes in regard to leg tremor as opposed to tremor of the upper extremity. Putnam describes two other types of operation. One of these is section of the lateral pyramidal tract in the spinal cord, usually at the second cervical segment. This procedure affects the leg as well as the arm; the resulting disability is in most instances slighter than that produced by ablation of the motor cortex, and the possible danger of convulsions from a cortical scar is avoided. Rigidity may be decreased. At the time Putnam wrote, this operation had been performed in seven cases; in every instance tremor was reduced and no deaths occurred. The operation had not been performed in cases of bilateral tremor. The other type of operation mentioned by Putnam, which, he states, has been successful in some cases, was proposed by Meyers and is aimed at subcortical structures, including the head of the caudate nucleus and the *ansa lenticularis*.

Before this discussion is brought to a close a digression should be made to draw attention to a recent report by P. C. Bucy, who was originally successful in operation for tremor.[1] He has described the surgical treatment of a man, aged twenty-three years, who suffered from severe tremor and mild spastic hemiparesis, involving the right extremities, from the age of eight years. The "arm" and "leg" areas of the posterior part of the precentral gyrus were removed. The tremor was completely abolished, but considerable paralysis of the hand and fingers resulted. Bucy's article is worthy of careful study apart from the result described—he discusses the different effects produced by extirpation of the cortical area and publishes some instructive illustrations.

To return to our original subject, we shall ask whether operation is worth while and, if so, what type of operation shall be performed. In regard to the first of these questions, it has to be remembered that the Parkinsonian syndrome includes many disabilities which may be present in varying degrees. As Putnam puts it, the relief of one disability may still leave the patient incapacitated. He also points out that the presence of mental deterioration or of an encephalitic psychosis may nullify any benefits of operation. It must be admitted that operation has been successful in certain cases, though the clinician, if he is wise, will be careful in accepting offhand the bald statement of a patient recovered from operation who holds that he has benefited from his ordeal. It will be readily admitted that a partially paralysed arm and hand would be preferable to an extremity whose movements could not be controlled. Which of the three types of operation mentioned by Putnam should be chosen may not be easy to determine. He thinks that they all owe their effect to encroachment on the pyramidal tract, and he also thinks that a cortical extirpation seems most in order for a patient suffering from a severe tremor of the left hand alone. Section of the lateral pyramidal tract, in his opinion, is indicated if the tremor involves both arm and leg. Bucy, in the case recently reported, has shown that cortical extirpation can be applied when the leg is involved. There is also an advantage in operation on the premotor cortex that the motor area can be accurately mapped out by electrical stimulation and the area in front of this, the premotor area, can then be removed with little risk that motor function will be affected. This, we understand, has been done in some cases in one Australian centre. When the condition is bilateral, operation becomes a most heroic procedure. The *British Medical Journal*, in commenting on this subject, declared[1] that little short of bilateral pyramidal section would be required to relieve the majority of the severe examples of *paralysis agitans* encountered in hospital practice, for unilateral severe Parkinsonism is uncommon. The general conclusion to be stated is that this subject is still in the early stages of its development. Patients who are carefully selected may be subjected to operation, but to quote Putnam once more, this will probably be a matter of trial and error, a minimal procedure being carried out at first with a reexploration should satisfactory relief not be obtained. Whatever is done, every detail must be observed, noted and recorded in every case that knowledge on the subject may accumulate.

[1] *Annals of Surgery*, December, 1945.

Current Comment.

EPITHELIOMA OF THE SKIN.

THE results of the treatment of epithelioma of the skin are of particular interest to us in Australia because of the very considerable incidence of the condition. The potential danger of skin cancer, the difficulty in educating the public in the best methods of prevention and cure,

[1] *British Medical Journal*, October 4, 1941, page 484.

and the excellent results that may be expected to follow correct and skilled treatment all make the subject of great importance. Joseph A. Elliott and David G. Welton have published a record of observations made on 1,742 patients seen over a 22-year period by the former author.[1] Only those suffering from cancer of the skin are included; lesions of the mucous membranes and malignant melanoma are excluded.

In the course of this article the whole subject is thoroughly traversed, and figures relating to results are prefaced by consideration of the ætiological factors, in particular those concerned with the role of pigment in the skin and the carcinogenetic effect of ultra-violet rays. The authors point out that there is ample support for the belief that the blond and ruddy complexions are disadvantageous from the point of view of protection from the harmful effects of ultra-violet rays, especially if they are also associated with a low degree of pigment formation. The relative insensitiveness of the Negro's skin is quoted in support, and surveys reinforce this evidence. Findlay's observations in cattle are also cited, in which he found that out of 170 cases of epithelioma discovered in examining one million cattle, all but three were sited in white areas of the hide. The work of dermatologists since the days of Unna on this aspect of skin cancer is summarized. Molesworth's special experience is referred to by these writers, and due prominence is given to the comparison he has made between the frequency of skin carcinoma in Great Britain and Australia, showing that the same stock when exposed to more intense solar radiation increase many fold their incidence of this neoplasm. In the discussion on the paper tribute was also paid to the work of both Paul and Molesworth in this field. Questions such as the exact mechanism by which these lesions are produced need not be pursued here, save that it seems certain that cellular changes occur which lead to a precancerous condition, and repeated irritation by the same physical agent induces definite malignant degeneration, this word being used in a loose and general way. Data are presented in this paper correlating the degrees of solar radiation in various geographical localities with skin cancer; these still further strengthen the argument. Occupation hazards did not penalize outdoor workers very heavily. They represented some 60% of the total, though, of course, there are other ways of inviting the attentions of the sun's rays besides working. The duration of the lesions when the patients first reported for treatment varied; the highest numbers were included under the periods of one to three years, but there is some evidence that education is improving this somewhat. Nearly 75% of the patients had a lesion not larger than one centimetre in diameter, and the commonest locations were the cheeks and the nose, and other parts of the face, which accounted in all for nearly 78% of the lesions. Of course, this is a truism; it is only placing further emphasis on exposure.

The type of lesion is of more than usual interest, and though details are given of the clinical types, it would be more significant if we could have histological confirmation. This is obviously not possible in many cases, for excision is now not often done, and biopsy is not always desirable unless there is doubt concerning the diagnosis. However, in 381 cases it was found that nearly half the specimens examined showed the growth to be of basal cell type, about 40% squamous celled, and the remainder of mixed variety. The method of treatment was thorough curettage, followed by electro-coagulation or desiccation, then by exposure of the area and the surrounding skin to fractional X radiation till a total of from 2,400*r* to 3,000*r* had been given. The authors followed up 1,052 patients for five years or more. Some 400 were followed by *questionnaires*; the remainder were seen personally. Five-year cures were obtained in 97% of these cases. The lesions were seen to heal satisfactorily in another 563, but the late results were not available. Recurrences and failures occurred in thirty cases, and three deaths took place. If the basis adopted for assessing the results of treatment is the total number of lesions treated and not the number of patients, the authors can record 58% five-year cures and 40% unaccounted for, as their 1,742 patients represented 1,928 carcinomata. Such surveys, made over a long period, stimulate interest, particularly in techniques of treatment. This is best left to the expert, but what should interest everyone is the matter of prevention. Everyone should know by now that the fair and lightly pigmented, especially those whose protective mechanism of pigment formation in the skin is poorly developed, are faced in the most literal sense with a hazard from unwise and constant exposure to solar radiation. The avoidance of this risk rests with themselves and their advisers.

[1] *Archives of Dermatology and Syphilology*, April, 1946.

PSYCHIC CAUSATION OF OBESITY.

THOUGH obesity is not such a perpetual reminder of the gaps in medical science as is the common cold, and is therefore not quite so humiliating to the profession, it can nevertheless evoke some awkward questions from the middle-aged whose weight is on the upgrade. The physician can, however, in defence take the position clearly announced by L. W. Newburgh that all persons afflicted with this condition are alike in at least one respect—they overeat.[1] In the opinion of one school adipose tissue can become pathologically affected and take on the characters of a malignant tumour. Others stress the dominating influence of the pituitary gland, pointing out that in a certain type of endocrine imbalance adipose tissue has first claim on the ingested food, no matter what the metabolic needs may be elsewhere in the body, and so the formation of fat is most difficult to avoid. Newburgh was at great pains to show that obesity is never directly produced by increased or diminished activity of an endocrine gland. In other words, no internal secretion is capable of so changing the metabolism that the total amount of fat in the body will increase unless the inflow of calories is greater than the outflow. He thought that it remained for the future to decide whether a hypothalamic disorder or other cerebral condition could cause obesity through abnormal stimulation of appetite. Dorothy Gill, of the Department of Nutrition, University Hospitals, Iowa, approaches the problem from the psychoanalytical aspect.[2] From the time of Julius Cæsar the difference between the contented and placid man of ample weight and the irritable, discontented and envious Cassius has been matter of everyday observation. But Dorothy Gill is concerned with the causation of this urge for overeating. Though her analysis has been confined to her own sex, many of her conclusions apply to the male as well. "In studying the obese person one must realize that food is more than an organic necessity to him; in all probability it constitutes his chief source of satisfaction for a multitude of interests." Without adopting Freudian concepts of erotogenous zones and attributing to babies psychic activities for which the appropriate cortical matter is totally undeveloped, we can agree that there is much to be said for the hypothesis of oral fixation involving direct oral gratification. Gill might have mentioned the well-known fact that smokers who suddenly give up the habit lay on weight; they find in nibbling confectionery an outlet for their oral urge. Some escape the grosser forms of weight increase by falling back on chewing gum. Natives of India often comment on the fact that white men are always doing something with their mouths; most smoke, but the obese have one dominant oral habit, they eat and eat. In this article it is contended that the obese woman has a home complex, is submissive and passive, is socially immature, is content with womanhood and has a lesser sexual libido. Here the author, however, does not clearly distinguish between what are results and what are causes and what are mixtures of the two, displaying that reciprocal reinforcement and mutual interplay so common in all organic processes. Still her insistence on oral gratification as a dominant psychic urge is a useful contribution to the vexed problem of obesity.

[1] *Archives of Internal Medicine*, Volume LXX, 1942, page 1033.
[2] *Journal of the American Dietetic Association*, May, 1946, page 398.

Abstracts from Medical Literature.

DERMATOLOGY.

Cutaneous and Subcutaneous Lesions of the Lower Limbs in Connexion with the Veins.

MAX WOLF (*Archives of Dermatology and Syphilology*, November-December, 1945) states that severe and permanent changes are to be expected as a consequence of long-standing varicose veins. Chronic stagnation not only interferes with the adequate nutrition of the affected areas, but also increases the tendency to recurrent phlebitis and inflammatory reactions in the perivenous tissues. The surface of an entire limb may become modified by ensuing atrophic or, more frequently, hypertrophic changes. Cutaneous lesions accompanying the "varicose symptom complex" may deviate from their usual clinical aspect, and difficulties may be encountered in their interpretation. One of the first signs of venous stasis, of increased capillary pressure and of capillary permeability may be the œdema of the dependent parts of the leg concomitant with pruritus. Itching is localized chiefly around the ankles, on the inside of the legs or on the shins. It increases toward evening after a day's work in an occupation that requires prolonged standing. Tiny papules are scattered among reddish discoloured areas of the lower extremities; some of the lesions are covered with blood-stained brownish crusts; frequent scratching facilitates the gradual transition to eczematization. Some phases of blood dyscrasias manifesting themselves as prurigo on the lower limbs may be difficult to differentiate from hæmostatic disturbances. Tiny itchy papules spread over a diffuse scaly dermatitis will not reveal the characteristics of leuchæmia, but repeated blood counts showing an increase of lymphocytes and a histological picture of an enlarged lymph node will afford sufficient evidence for a diagnosis of *prurigo lymphatica*. Another misleading sign in association with stasis is the occurrence of intracutaneous petechiæ and pigmentations. Pinpoint-sized to pinhead-sized reddish spots changing to greenish brown and finally to dark brown usually form discrete nummular patches on all parts of the leg, but mostly on the lower third. These ill-defined lesions have a superficial resemblance in distribution and appearance to Schamberg's progressive pigmentary dermatosis and to the pigmented purpuric lichenoid dermatitis of Blum and Gougerot. Eczematous conditions and ulcer formation are the outstanding features of the varicose state; phlebitis and periphlebitis may precipitate their development. The different stages of varicose eczema are characterized by branny scaling, or flake-like exfoliation alternating with moisture and crust formation. Bacteria and fungi find an excellent medium for propagation in the permanently engorged legs. Dermatophytosis between the toes or of the nails serves as a source of repeated reinfection of the limbs. Secondary infections by streptococci in the form of lymphangitis or attacks of recurrent erysipelas may be followed by enormous swelling of the legs. Even without these infections the chronic inflammation of the superficial layers of the skin may slowly spread to deeper parts of the cutis and to the subcutis and may produce hyperplastic tissue changes. The state of pseudo-elephantiasis may be accompanied with verrucous or papillary projections of skin. Patients susceptible to atopic eczema may demonstrate the topographic coincidence of neurodermatitis with varicosities. In its early stage neurodermatitis of the limbs may be cured by elimination of incompetent feeding veins. *Lichen chronicus simplex* (Vidal) may also exhibit the close connexion with varicose veins. *Prurigo nodularis*, a severer variant of neurodermatitis, may develop as a final result of intense scratching caused by hæmostasis. Hypertrophic, hard, extremely itchy, tumour-like nodules with a verrucous surface are found close to varicose veins on a generally lichenified base. With the treatment of surrounding varicosities these nodules may occasionally diminish in size or may even disappear. Perivenous pachyderma or dermatosclerosis is a special type of inflammatory process which starts insidiously in the periphery of smaller, more deeply seated varicosities. The inflammation becomes visible by the establishment of a brownish, indurated, palm-sized plaque on the leg above the inner side of the ankle, which may last indefinitely, with gradually increasing tenseness. The area may change its colour to dark brown; it may become depressed below the surrounding surface by fixation to the subjacent tissues, and the inflammation may advance to the anterior and lateral aspect or may even continue to the upper third of the leg. Cuff-like constrictions of sclerotic skin are characteristic features of this disease. Periostitic thickenings similar to those in tertiary syphilis may be perceptible. The sclerotic atrophic tissues are liable to break down and form chronic ulcers.

Undecylenic Acid in the Treatment of Dermatomycosis.

A. L. SHAPIRO AND S. ROTHMAN (*Archives of Dermatology and Syphilology*, September, 1945) state that a common error in the management of dermatomycosis is the application of the strongest possible fungicide agent. The general practitioner often misjudges the irritability of the skin as well as the degree of disinfecting power required for treatment. The requirements of a suitable fungicide preparation are: (i) It should be able to kill the particular fungi. (ii) It should be able to penetrate to the particular location of the fungi. (iii) It should not irritate the skin in the particular phase of the disease in which it is applied. A new therapeutic principle was introduced in 1938 by Peck and Rosenfeld, who found that organic fatty acids occurring in sweat have considerable fungicidal action without any irritating effect. They found that the sodium salt of undecylenic acid has the strongest fungicidal action of all tested fatty acids and their salts. The authors for the first two years tested a preparation consisting of zinc undecylenic 20% and undecylenic acid 5% in a vanishing emulsion base. Complete clinical cure could be achieved with the undecylenic ointment within four weeks for the great majority of patients with *dermatophytosis pedis*, regardless of the species of causative fungi. This result does not indicate that the new preparation is far superior to older standard preparations, particularly to the sulphur-salicylic ointments. However, the undecylenic ointment is less irritating than any other preparation of similar fungicidal strength. Where tincture of iodine and sulphur salicylic ointment would be risky for patients with extended vesiculation, erosions, rhagodes and œdema of the feet, undecylenic ointment can be used without the slightest danger of irritation. In the treatment of *tinea cruris* and *axillaris* complete cure was accomplished easily without irritation by the use of undecylenic ointment. Against *tinea circinata* the results were not so satisfactory. Infection with *Microsporon audouini* on the glaborous skin of adults and children responded extremely well. In the treatment of onychomycosis the authors' results were not encouraging.

Dermatitis Venenata following the Use of Penicillin Ointment.

LEONARD SIMPSON MARKSON (*Archives of Dermatology and Syphilology*, November-December, 1945) reports that it has been previously noted that penicillin when administered parenterally gives rise to varied cutaneous manifestations, such as urticaria, recurrent vesicular eruptions and exfoliative dermatitis. A case of contact dermatitis has been reported in a medical officer who was preparing penicillin. To these cases of penicillin sensitivity the author adds a case in which *dermatitis venenata* developed on the eyelids and face of a white man following treatment for mild conjunctivitis with penicillin ointment. A report of the case appears.

Dermatitis due to a Case-Hardening Compound.

G. P. B. WHITWELL (*The British Journal of Dermatology and Syphilis*, September-October, 1945) writes about a dermatitis due to a process in which organic carbonizers are mixed with chemicals. The author has seen two main types of such products used. One is wood charcoal impregnated with 5% sodium carbonate; the other is wood charcoal impregnated with barium carbonate. In both cases the carbon pellets are manually packed round the components to be hardened in metal pots. After being in the furnace the pots are cooled and emptied into a sieve, which is used to free the components of carbon. The process of packing and unpacking the pots entails much soiling of the skin, and the dust tends to get through and over the tops of the gloves. Severe dermatitis is seen among the work-people. The rash is eczematoid and mainly affects the backs of the hands and the flexor surface of the forearms, the face being sometimes involved. The rash often evolves rapidly, especially when the man remains at work after an erythema has started. Although the rash is eczematoid, a striking feature of an early case is that the rash can be seen to originate from the follicles; this is especially noticeable in the flexor aspects of the forearms. The cause of the rash is undoubtedly the sodium carbonate in the carbon pellets. Even when the carbon is washed after use

it is still strongly alkaline to litmus. Carbon containing barium was not considered so harmful. The management of this form of dermatitis begins with the new employee, who should be warned to report any rash immediately. Men who washed frequently during work were more liable because with a damp skin on his arms a man soon had a strong solution of soda on his skin. The author is doubtful whether barrier creams prevent this type of dermatitis.

UROLOGY.

Primary Carcinoma of the Ureter.

J. A. LAZARUS AND M. S. MARKS (*The Journal of Urology*, August, 1945) recall that in 1934 the first-named author, after careful search of the literature, was able to collect only 68 cases of primary carcinoma of the ureter including three of his own. Since then 115 additional cases have been reported. It is considered that this reflects an increasing vigilance on the part of urologists rather than increased incidence. About one-half of the tumours are in the lower segment; one-half are in the upper; and in 46% of all cases the renal pelvis is dilated. These tumours often tend to occlude the duct. About 42% were of non-papillary type. The greatest prevalence of the disease was in the sixth and seventh decades of life. These carcinomata show a great invasion and metastasizing tendency, particularly to regional lymph nodes, the liver and bones. Hæmaturia was found to be the outstanding symptom, being present in about 70% of cases. Pain and the presence of an enlarged kidney are the other important signs. The real pathognomonic sign of this disorder is a clear-cut and persistent filling defect in the ureterogram, often associated with ectasia of the ureter and pelvis above it. This disease would be less often missed if repeated ureterograms were made in all cases in which manipulation of a ureteric catheter in order to pass an obstruction causes bleeding through the catheter, and in all cases in which the cause (calculus or otherwise) of a ureteric obstruction is not satisfactorily demonstrated. The most suitable treatment is complete extraperitoneal nephro-ureterectomy. A review of the literature has satisfied the authors of the dangers of incomplete urological surveys of so-called minor urological complaints.

Thrombosis and Thrombophlebitis of the Renal Veins.

B. S. ABESHOUSE (*Urologic and Cutaneous Review*, November, 1945) has collected from the literature 228 cases of renal vein thrombosis and states that the condition is relatively uncommon. Many cases are probably encountered, however, and are not recognized. The condition occurs at any age and occurs equally in both sexes; it is slightly more often unilateral than bilateral. The primary type, which originates in the renal venous system, occurs usually in infants and children and is associated with ileo-colitis. The secondary type, which is the result of extension of thrombosis from the peripheral veins or the inferior *vena cava*, is seen usually in adults following pregnancy, surgical operations or recumbency. There are also described suppurative and non-suppurative types. Occlusion of the main renal vein leads to total renal necrosis with hæmorrhagic infiltration in and around the kidney. Occlusion of a branch of the main vein leads to the formation of a wedge-shaped hæmorrhagic infarct. The clinical features are not constant, but the possibility of this condition should be borne in mind when any of the following signs or symptoms are present: (*a*) sudden onset of sharp pain in the kidney area with localized tenderness and subsequent palpation of an enlarged kidney; (*b*) fever, leucocytosis and prostration; (*c*) gross or microscopic hæmaturia and albuminuria followed by progressive oliguria or anuria.

Congenital Renal Anomalies in the Army.

J. H. HARRISON AND T. W. BOTSFORD (*The Journal of Urology*, April, 1946) state that men are often accepted into military service without discovery of congenital renal anomalies because of the absence of symptoms and since special urographic study is not available. However, symptoms sometimes appear following the exigencies of military service. Excessive physical exertion, trauma, exposure to unusual climatic conditions and intercurrent infections are contributory factors to the appearance of symptoms. Congenital anomalies of the kidney are numerous and relatively common, but the anomalous organ is more susceptible to disease than the normal organ only if the anomaly interferes with the circulation or the excretory function of the kidney. Many anomalies do not interfere with the functioning of the organ and may not therefore contra-indicate full physical activity. However, severe abnormalities of form and structure, complicated by disturbance of dynamics, superimposed infection, calculus or hæmorrhage, are incompatible with active military service. The authors analyse 72 cases of congenital renal anomalies occurring in patients admitted to a United States general hospital in the South-West Pacific Area. They were found among 29,000 hospital admissions, which is a lower incidence than that found in the usual civilian population. Twenty-one of these patients required surgical operation; seven operations were for calculi, the other fourteen for congenital hydronephrosis. Forty patients were returned to full military duty, six to limited duty, and twenty-five were evacuated to the United States.

Complete Rupture of the Membranous Urethra.

W. F. LEADBETTER (*The Journal of Urology*, December, 1945) describes a technique to restore the position of the prostate and to secure better urethral apposition in cases of complete rupture of the membranous urethra. In these cases the prostate and bladder base are sometimes dislocated upwards. When the bladder is opened for deviating cystostomy the prevesical space is cleared of all clots and a number 18 F Foley bag retention catheter is passed from the external meatus along the urethra and then threaded through the prostatic portion of the canal and on into the bladder. Its bag is inflated and tension is made on the catheter. To try to depress the prostate into its normal position a mattress suture of heavy silk is passed through each lateral lobe of the gland and is tied in the perineum through rubber tubing. No attempt to repair the urethra is made. The prevesical space is well drained with at least two drains passed down to the prostate. Even if a stricture does occur after the urethra heals, secondary perineal operation can be done with greater chance of success than if the prostate had not been replaced into a fairly normal position.

Sclerosing Injection of Polycystic Kidneys.

W. W. YOUNG (*The Journal of Urology*, April, 1946) states that, while the use of sclerosing solutions in lieu of excision is generally losing popularity in other fields of surgery, its application to polycystic kidneys to obliterate larger cysts and so to conserve functioning renal tissue offers at least theoretical advantage. A young female patient with rapid unilateral polycystic growth was clinically much improved. Although only one kidney was treated, her approach toward the borderline of nitrogen retention was noticeably retarded. This patient has since been able to bear a living child and has passed easily through the dangerous complication of an acute pyelonephritis. After syringe evacuation of the larger cysts, 5% sodium morrhuate solution is injected by 1·0 millilitre for every 10·0 millilitres of cyst fluid evacuated. Some of the larger cysts held up to 70 millilitres. Many of the deep-seated cysts were located by palpation, though not discernible on the surface. The true cyst fluid was turbid and greyish-brown in colour. To protect the renal pelvis from accidental infection, an intravenous injection of 5·0 millilitres of 8% indigo carmine solution is given before the patient leaves bed, and again after anæsthesia is complete. If a needle taps the pelvis, the aspirated fluid is essentially clear, but pale green in colour. A good supply of syringes, ranging from 10 to 50 millilitres in size, is used and plenty of needles ranging from 22 to 17 gauge are put out. After such treatment the kidney shrinks to nearly one-half its previous size, and the surface becomes flabby and loose. The surface incision is closed with drainage, the drains being removed on the fourth day.

Renal Calculi Associated with Hyperparathyreoidism.

E. N. COOK AND F. R. KEATING (*The Journal of Urology*, December, 1945), as a result of a deliberate search for the disease, have discovered eighteen cases of hyperparathyreoidism in the Mayo Clinic in eighteen months. In thirteen of these cases, renal calculi were the presenting symptoms, and in the majority of this calculous group disease of bone was slight or absent. In the same period of eighteen months, hyperparathyreoidism was present in 2% of all cases of renal calculi; but only a small proportion of patients with renal stones were carefully investigated with this diagnosis in mind. The authors therefore feel that the observations of Albright and his associates deserve reemphasis, namely, that hyperparathyreoidism is commonly associated with renal calculi and is an important ætiological factor.

Bibliography of Scientific and Industrial Reports.[1]

THE RESULTS OF WAR-TIME RESEARCH.

During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided that a large proportion of the results of this research should now be released for general use.

The United States Department of Commerce, through its "Publication Board", is now issuing abstracts of these reports in the form of a "Bibliography of Scientific and Industrial Reports". The complete bibliography is being received in Australia, and extracts likely to be of interest to readers of this journal will be reproduced as far as practicable each week.

The original reports may be obtained in two ways: (a) Those marked with an asterisk may be obtained by approved applicants without cost on making application to Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies are available for reference in public libraries. (b) In other cases microfilm or photostat copies of the original report may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Readers desiring to avail themselves of this service should send the Australian equivalent of the net United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.E.2. All other charges will be borne by the Council for Scientific and Industrial Research.

Further information on the subjects covered by the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division, Department of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.

FOLKERS, KARL, PHILLIPS, RICHARD F., AND SHUNK, CLIFFORD H. Studies on the preparation of non-volatile toxic agents, to December 31, 1943. (OSRD Report 4586.) Off. Pub. Bd., Report, PB 5905. 1945. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report describes the preparation of members of three classes of non-volatile compounds, to be tested for toxicity by subcutaneous injection in mice. The most important discovery was in the class of urethan derivatives of pyrocatechol, resorcinol, hydroquinone, pyrogallol and related compounds. Pyrocatechol bisdimethylurethan was found to be very toxic to mice (LD_{50} 1·4 mg./kg., subcutaneous). This finding indicates the importance of the urethan group in contributing to the toxicity of physostigmine analogs, which contain both urethan and quaternary ammonium or tertiary amine groups. Derivatives of bisapomethylbrucine and the nitrogen mustards were also prepared. Formulæ of compounds and results of toxicity tests are listed in Tables I, II and III. Table IV presents experimental data on the new compounds prepared. This is a final report under contract with Merck and Company.

HOGEBOOM, GEORGE H., *et alii*. I. The necrotizing action of certain substances related to mustard gas, H, or to the nitrogen mustards. II. A comparison of the vesicant action exerted on human skin by mustard gas, H, and by mixtures of H with wetting agents or solvents, to March 1, 1945. (OSRD Report 4852.) Off. Pub. Bd., Report, PB 5491. 1945. 29 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Most of the compounds tested were sulphonium salts, sulphones or sulphoxides. Their necrotizing activity on rabbit skin in terms of H or of parent nitrogen mustard is briefly discussed and tabulated as well as their activity in various solvents. In Part II are reported results of a few trial contacts made on heavily sweating human volunteers. This is a progress report of the Rockefeller Institute for Medical Research.

HAMILTON, C. S., *et alii*. Organic arsenicals and other toxic agents, to September 1, 1944. (OSRD Report 4533.) Off. Pub. Bd., Report, PB 5954. 1945. 67 pp. Price: Microfilm, $1.00; Photostat, $5.00.

This progress report under contract with the University of Nebraska gives complete experimental details and references. Included are: (1) preparation of ten arsenicals for toxicity tests; (2) preparation of three mercury, one chromium and seven sulphur compounds for toxicity tests; (3) discontinued problems on the toxic agent programme; (4) preparation of nineteen derivatives of chemical warfare agents in 100 g. lots, and checking twenty procedures for preparing derivatives developed at the CWS-MIT Laboratory.

GAGGE, A. P. Biophysical experimentation in flight in Constellation (C-69). (Army Air Forces. Aero Medical Laboratory. ENG-49-695-29-E, Add. 1.) Off. Pub. Bd., Report, PB 6956. 1944. 2 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report serves as an amendment to Memorandum Report ENG-49-695-29E. It is pointed out that an oxygen system is essential in all pressure cabin aircraft for emergency use under the following conditions: when weather conditions prevent an immediate descent; when air evacuation patients are carried; and when older passengers are carried in whom anoxia can precipitate a heart attack because of latent cardiac disease. See also PB 6955 on page 497.

HEATON, C. D., TORBOHN, E. W., AND NOLLER, C. R. The preparation for toxicity tests of some ethylene and acetylene derivatives, to September 1, 1944. (OSRD Report 4325.) Off. Pub. Bd., Report, PB 5934. 1944. 40 pp. Price: Microfilm, 50c.; Photostat, $3.00.

Because of the high toxicity of methyl acetylenedicarboxylate, 33 derivatives of ethylene and acetylene were prepared and submitted for toxicity tests. Nine of the compounds showed toxicity by inhalation at concentrations below 3 mg./1. 1-Methoxy-2-pentyn-4-one was the most toxic compound of the series and showed an approximate LC_{50} of 0·6 mg./1. Sixteen of these compounds were tested for toxicity by subcutaneous injection in mice, but none of them showed significant toxicity. Bromopropiolic acid and 3-iodo-2-propyn-1-ol were vesicant on application of 100 microgrammes. The methyl-, ethyl- and 2-chloroethyl esters of propiolic acid caused intense lacrimation. Data on toxicity are given in Table I. Twenty-four references. This is a progress report under contract with Stanford University.

KARNOFSKY, DAVID, *et alii*. Progress report on "the clinical pathology of di (β-chloroethyl)-methylamine (TL 146)". (OSRD Report 1339.) Off. Pub. Bd., Report, PB 5936. 72 pp. Price: Microfilm, $1.00; Photostat, $5.00.

This progress report, under contract with New York University, is based upon informal monthly progress reports from July, 1942, to date. The authors have endeavoured to cite all pertinent investigations. Eighty-six references are given. The study gives in detail the effects of TL 146 when administered by inhalation or by the injection of fresh solutions of the hydrochloride salt. The original observations reported herein have been confined to small animals (mice, rats and rabbits), though where data are available reference is made to the effects of the compounds in other species. Toxicity data and the chief clinical manifestations in relation to the dose and route of administration of TL 146 are summarized in Table I. Other tables and photomicrographs are also given.

LINTHICUM, E. S., AND ELDRIDGE, W. A. Toxicity of white phosphorous smoke. (Edgewood Arsenal Technical Report 59.) Off. Pub. Bd., Report, PB 6265. 1931. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Laboratory and field tests were made to determine the effects of white phosphorus smoke upon laboratory animals. A field test was run wherein four goats and nine rabbits were exposed for 55 minutes to a dense smoke caused by burning 150 pounds of white phosphorus over a small area. A slight serous nasal discharge was the only effect indicating that white phosphorous smoke is non-toxic in the highest field concentrations for an hour's exposure.

LOVELACE, W. RANDOLPH, II. Physiology of continuous and intermittent positive pressure respiration. (Army Air Forces. Engineering Division. ENG-49-695-1U.) Off. Pub. Bd., Report, PB 6929. 1944. 51 pp. Price: Microfilm, $1.00; Photostat, $4.00.

Description of experiments on anæsthetized dogs before and after disturbance of the circulation produced by acute hæmorrhage, intestinal manipulation and hind-limb trauma. This is a serial report under contract with the Mayo Clinic. Illustrations and tables are included.

MAISON, GEORGE L., MAASKE, CLARENCE A., AND MARTIN, ERNEST E. Description of the Materiel Command human centrifuge, techniques employed therewith and results of studies on normal "G" tolerance of human subjects. (Army Air Forces. Engineering Division. ENG-49-696-4D.) Off. Pub. Bd., Report, PB 5144. 1943. 20 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This human centrifuge is a well-equipped and versatile instrument which allows for accurate study of man's reactions and resistance to "g" in normal and "protected" conditions. The highest "g" values the average young male can withstand have been determined. A recording signal system is an accurate and inexpensive method of objectively determining man's visual and auditory functions and reaction

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

time while exposed to radial acceleration. A description and illustrations of the centrifuge are attached as appendix 1. A standard technique of operation has been developed and is described in appendix 2. Appendix 3 is concerned with normal "g" tolerances and includes graphs and a table.

ROTARIU, GEORGE J., *et alii*. The toxicity and pathology of phosgene oxime (dichloroformoxime) to March 31, 1945. (OSRD Report 5002.) Off. Pub. Bd., Report, PB 5902. 1945. 7 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Special precautions were taken to test a sample before spontaneous deterioration had occurred. The results reveal that it is less toxic than either phosgene or mustard for various animal species; the mouse 10-minute LC_{50} is 0·7 mg/l. and other tested species are more resistant. The pathological changes are quite different from those produced by CG: they resemble those produced by H. Toxicity data are tabulated. This is a progress report under contract with the University of Chicago.

ROUILLER, CHARLES A. First-aid kit for chemical warfare service plants. Final report on Project D 5.1-1. (Chemical Warfare Service. Edgewood Arsenal Technical Report, E.A.T.R. 291.) Off. Pub. Bd., Report, PB 6336. 1938. 10 pp. Price: Microfilm, 50c.; Photostat, $1.00.

A kit was developed, a specification was written, and twenty of the kits were procured from industry under this specification and service tested. After the kit had been revised to correct defects, it was decided that the military requirements for first-aid facilities in chemical warfare service plants can best be met by including in the equipment of each plant materials of the kind and quantity required by the specific operations conducted therein. Therefore the project was closed.

SMITH, LEE IRVIN, AND ENGLEHARDT, VAUGHN. Paraphenylenediamine compounds, to February 10, 1944. (OSRD Report 5247.) Off. Pub. Bd., Report, PB 5937. 1945. 31 pp. Price: Microfilm, 50c.; Photostat, $3.00.

This report describes investigations on forty-three compounds which were tested appropriately for toxicity by injection and inhalation. These compounds have been classified into eight groups for presentation of the data. One group of these compounds, the N,N′-alkyl-phenylenediamines, produced severe erythema when tested as vesicants and showed moderate toxicity by inhalation. The most toxic methohalide of the phenylenediamines was N,N,N′, N′-tetraethyl-p-phenylenediamine methiodide which showed an LD-50 of approximately 10 mg./kg. The hydrochlorides of various *p*-phenylenediamines showed an LD-50 of approximately 20 mg./kg. Unless otherwise stated, the toxicity data in the tables are for mice—ten-minute exposure—ten-day observation. This is a progress report under contract with the University of Minnesota.

SWEENEY, H. M., AND JOFFE, MILTON H. Biophysical experimentation in flight in Constellation (C-69). (Army Air Forces. Aero Medical Laboratory. ENG-49-695-29E.) Off. Pub. Bd., Report, PB 6955. 1944. 23 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Data reported were obtained on April 15, 1944, June 2, 1944, and June 12, 1944, during flights of two, six and eight hours' duration, respectively, with 44 unselected personnel aboard a C-69 airplane. The tests were made to assay the effect of rapid decompression, to study the changes in temperature and percentage of relative humidity in the pressurized compartment, and to measure the oxygen and carbon dioxide content of the air in the pressurized compartment. Appendices give photographs, records, results and tables. See also PB 6956 on page 496.

WOOSTER, H. A., AND MILLER, D. S. The toxicity to white rats of red squill powder and various fractions isolated from it. (University of Chicago Toxicity Laboratory Special Report. Chemical Warfare Service Contract W-49-057-cws-23.) Off. Pub. Bd., Report, PB 5522. 1945. 15 pp. Price: Microfilm, 50c.; Photostat, $1.00.

In the course of developing a chemical assay method for red squill powder, Dr. C. R. Noller submitted 34 fractions isolated from red squill to this laboratory for toxicity testing. These 34 fractions were tested on 2,272 rats of several strains and both sexes. The results of the tests on 1,188 of these animals, males of the Maquran strain, are given in this report. Red squill powder was found to have an LD-50 of 458 mg./kg. within the fiducial limits of 507-414. A table of fractions found to be most toxic is given.

BERGMANN, MAX, *et alii*. Some reactions of mustard gas with proteins and proteolytic enzymes. (OSRD Report 1439.) Off. Pub. Bd., Report, PB 6805. 1943. 47 pp. Price: Microfilm, 50c.; Photostat, $4.00.

The extensive studies described in this report were performed in order to gain further insight into the action of mustard gas under proteins, proteolytic enzymes, and other constituents of biological systems. Since water is a major component of biological systems, some transformations of mustard gas in water are also discussed. The experiments were performed largely with redistilled thiodiglycol mustard gas (DH). In a few experiments, Levinstein mustard gas (HS) was employed. Tables and equations are included. This is a formal progress report under contract with the Rockefeller Institute for Medical Research.

GEILING, E. M. K., AND MCLEAN, FRANKLIN C. Report on "techniques employed in toxicity determinations at the University of Chicago Toxicity Laboratory, to August 24, 1942". (OSRD Report 893.) Off. Pub. Bd., Report, PB 6576. 1942. 70 pp. Price: Microfilm, $1.00; Photostat, $5.00.

This is a report of methods in current use at the Toxicity Laboratory. In addition to the description of the apparatus, an attempt has been made to evaluate the advantages and disadvantages of the various methods. The report discusses methods for studying toxicity of vapours; the Benesch machine; constant flow of micro-apparatus for exposure of mice to volatile toxic agents under controlled conditions; smoke programme; techniques used in the investigations of the pathological changes produced in animals by chemical warfare agents; methods used in examining animal eyes for toxicity data; methods used in testing skin irritants on mouse and man; maintenance, including quarters and diet, of aimals for toxicity determination; statistical treatment of data. Report contains photographs and diagrams of equipment and tables of results. Progress report under contract with University of Chicago.

GEILING, E. M. K., AND MCLEAN, FRANKLIN C. Report on "the toxicity of β-chlorethylthiocholine-chloride". (OSRD Report 276. University of Chicago Toxicity Laboratory Report No. 2.) Off. Pub. Bd., Report, PB 5562. 1941. 13 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This substance was synthesized with the idea of combining the toxicity of a quaternary base with mustard. The report gives descriptions of experiments revealing its irritative power to the human arm and the rabbit eye, the systemic toxicity to mice and rats (including in the latter the toxicity for application to the skin), and the effect on the circulation of the cat. It was not sufficiently irritating to be considered toxicologically important. Tables are included. This is a progress report under contract with the University of Chicago. The experimental work was done by Julius M. Coon and Jules H. Last.

KOONTZ, A. B., AND WILKES, A. E. The use of Turkey red oil and other agents for the removal of liquid mustard from the skin. (Chemical Warfare Service. Edgewood Arsenal Technical Report, E.A.T.R. 30.) Off. Pub. Bd., Report, PB 6295. 1931. 39 pp. Price: Microfilm, 50c.; Photostat, $3.00.

The objects of this work were (1) to determine the efficiency of Turkey red oil as an agent for the removal of liquid mustard from the skin, and (2) to compare the efficiency of Turkey red oil with that of kerosene and alkalis for the removal of liquid mustard from the skin. Test results indicated that, if liquid mustard is removed by Turkey red oil within the first few minutes after its application, either no burn or a very slight burn results. Some diminution in the size of the burn is accomplished if the mustard is removed by Turkey red oil as late as thirteen minutes after application. It is recommended that consideration be given to the development of Turkey red oil with a view to the replacement of kerosene and sodium bicarbonate in first-aid treatment. Separate consideration should be given to the desirability of employing Turkey red oil in plants where mustard gas is manufactured or handled. Includes references and photographs of experimental subjects.

LAZIER, W. Z. Progress report on "CMR-NDRC joint project on BAL ointment. Report on the work of the pharmaceutical and chemical subcommittee for the period of January through April, 1943, to June 23, 1943". (OSRD Report 1652.) Off. Pub. Bd., Report, PB 6816., 1943. 84 pp. Price: Microfilm, $1.00; Photostat, $6.00.

This is a progress report under contract with E. I. du Pont de Nemours and Company, which in conjunction with the final report of the Subcommittee on Vehicles and Packages Suitable for BAL Ointment or Jellies for the Armed Forces (included here), constitutes the final report of the subcommittee as a whole. Fifteen formulations of ointments, creams and jellies containing 5% BAL were selected, modified to improve consistency and increase stability, prepared and studied experimentally. The standards and methods for accelerated aging tests, for determining physical properties at high and low temperatures, for chemical analyses, for packages and for determining the effects of the ingredients on the package, were fixed. Standards and methods for the biologic testing and for the determination of comparative efficacy of fresh and aged preparations in treatment of Lewisite burns of the eyes and of the skin were also fixed. Selected formulæ were tested for various special effects. Tables, graphs and diagrams are provided.

British Medical Association News.

NEW SOUTH WALES BRANCH NEWS.

THE following books, pamphlets *et cetera* have been added to the library of the New South Wales Branch during the period May 1 to August 22, 1946.

Alexander, F. M.: "The Use of the Self", 1931.
Alexander, F. M.: "Constructive Conscious Control of the Individual", Second Edition, 1924.
American Medical Association: "Voluntary Prepayment Medical Care Plans", 1946.
Arnold-Forster, H. O.: "The Coming of the Kilogram", 1898.
Association for Research in Nervous and Mental Disease: "Trauma of the Central Nervous System", 1945.
Australia, Commonwealth of: "Australian Housing", Bulletin Number 9, July, 1946. Department of Health: "School Canteens", by W. M. Stenhouse, 1946. "Food Consumption Levels in Australia and the United Kingdom", 1945. National Health and Medical Research Council Nutrition Committee, Special Report No. 1: "The Food Consumption", 1945.
Bankoff, George: "Plastic Surgery", 1943.
Beck, Samuel J.: "Rorschach's Test. II. A Variety of Personality Pictures", 1945.
Bethea, Oscar: "1945 Yearbook of General Therapeutics."
Bibby, Cyril: "Sex Education", 1946.
Brook, Charles: "Battling Surgeon", 1945.
Cope, Zachary: "Early Diagnosis of the Acute Abdomen", Ninth Edition, 1946.
Dunlop, D. M., *et alii*: "Textbook of Medical Treatment", Fourth Edition, 1946.
Elderton, W. P. and E. M.: "Primer of Statistics", 1910.
Everett, Houston S.: "Gynæcological and Obstetrical Urology", 1944.
Garceau, Oliver: "Political Life of the American Medical Association", 1941.
Geschickter, C. F.: "Diseases of the Breast", Second Edition, 1945.
Great Britain—Medical Research Council: Special Report Number 235: "Chemical Composition of Foods", by R. McCance and E. Widdowson, Second Edition, 1946. War Memorandum Number 17: "Environmental Warmth and its Measurement", by T. Bedford, 1946.
Greenhill, J. P.: "1945 Yearbook of Obstetrics and Gynæcology."
Hale-White, W.: "Materia Medica", revised by A. H. Douthwaite, Twenty-Sixth Edition, 1946.
Hawkins, R. R.: "Scientific, Medical and Technical Books Published in the United States of America", 1930-1944.
Hewer, C. Langton: "Recent Advances in Anæsthesia and Analgesia", Fifth Edition, 1946.
Jackson, C. and C. L.: "Diseases of the Nose, Throat and Ear", 1945.
Kovács, Richard: "Electrotherapy and Light Therapy", Fifth Edition, 1945.
Levine, Maurice: "Psychotherapy in Medical Practice", 1944.
Luck, James Murray: "1946 Annual Review of Physiology."
Maxwell, James: "Introduction to Diseases of the Chest", Second Edition, 1944.
Medical Correspondence College: "Guide to Higher Appointments for Nurses"; "Guide to Medical Examinations"; "Hints on Writing a Thesis for the M.D. Degree"; "How to Obtain the Diploma in Psychological Medicine"; "How to Pass the C.M.B. Examination"; "How to Pass the Conjoint Board Examinations"; "How to Pass the State Registration Examinations"; "The M.D., London"; "The M.R.C.P. and How to Obtain it".
Painter, Charles F.: "1945 Yearbook of Industrial and Orthopædic Surgery."
Phillips, R. A., *et alii*: "Copper Sulphate Method for Measuring Specific Gravities of Whole Blood and Plasma", 1945.
Piney, A., and Wyard, S.: "Clinical Atlas of Blood Diseases", Sixth Edition, 1945.
Price, Weston A.: "Nutrition and Physical Degeneration", 1945.
Radio Doctor, The: "Good Health, Children!", 1944.
Rafferty, T. N.: "Artificial Pneumothorax in Pulmonary Tuberculosis", 1945.
Reese, H. S.: "1945 Yearbook of Neurology, Psychiatry and Endocrinology."
Scott, George Dow: "Heredity, Food and Environment in the Nutrition of Infants and Children", 1942.
Simmons, G. H., and Fishbein, M.: "Art and Practice of Medical Writing", 1925.
Sulzberger, M. B., and Baer, R. L.: "1945 Yearbook of Dermatology and Syphilology."
Sutton, R. and R.: "Diseases of the Skin", Tenth Edition, 1939.
Topley, W., and Wilson, G. S.: "Principles of Bacteriology and Immunity", Third Edition, 1946.
Underwood, E. Ashworth: "Manual of Tuberculosis", Third Edition, 1945.
University of Sydney Medical School: "Yearbook for 1945-1946."
Periodicals: *British Journal of Pharmacology and Chemotherapy*, Volume I, 1946; *Journal of Bacteriology*, Volume LI, 1946; *Proceedings of the Royal Australasian College of Physicians*, Volume I, 1946; *Thorax*, Volume I, 1946.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioner has been released from full-time duty with His Majesty's Forces and will resume practice as from the date mentioned:

Dr. Alexander Owen, c.o. Dr. B. P. Anderson Stuart, British Medical Association House, 135, Macquarie Street, Sydney (September 1, 1946).

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 150, of August 15, 1946.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

SX3256 Lieutenant-Colonel (Temporary Colonel) F. N. Le Messurier, D.S.O., relinquishes the rank of Temporary Colonel and is transferred to the Reserve of Officers (Australian Army Medical Corps) with the rank of Lieutenant-Colonel and is granted the rank of Honorary Colonel, 23rd May, 1946.

VX39709 Lieutenant-Colonel J. B. D. Galbraith is transferred to the Reserve of Officers (Australian Army Medical Corps), 17th May, 1946.

CASUALTIES.

ACCORDING to the casualty list received on August 19, 1946, Captain J. F. R. Burnett, A.A.M.C., of Perth, who was previously reported missing, is now reported "presumed to be dead".

Medical Practice.

FRIENDLY SOCIETY LODGE PRACTICE IN VICTORIA.

THE following letter was received by the Medical Secretary of the Victorian Branch from the Acting Prices Commissioner on the subject of capitation fees payable to medical officers by friendly societies. It is published at the request of the Medical Secretary.

[COPY.]

COMMONWEALTH OF AUSTRALIA.

Department of Trade and Customs.

Telephone: F 9471.
Ref. No.: V.P.40/1144.

Victorian Prices Branch,
Temperance and General Buildings,
143-7 Collins Street,
Melbourne, C.1,
9th August, 1946.

Dear Sir,

In furtherance of my letter dated 21st November, 1944, on the subject of capitation fees payable to medical officers by

the friendly societies, I append hereunder details of the complete scale of maximum fees, as approved by the Commonwealth Prices Commissioner:

Rates Per Annum.	Within the Melbourne Metropolitan, Ballarat, Bendigo and Geelong Areas.	Elsewhere in Victoria.
Male members	26s.	32s.
Female members (except I.O.R. and O.S.T.)—		
(*a*) of male lodges	26s.	32s.
(*b*) of female lodges—		
(i) without dependants	15s. 7d.	15s. 7d.
(ii) with dependants ..	23s. 5d.	23s. 5d.
Female members of I.O.R. and O.S.T.—		
(*a*) in mixed tents—		
(i) without dependants	20s. 10d.	20s. 10d.
(ii) with dependants ..	26s.	32s.
(*b*) in female tents—		
(i) without dependants	15s. 7d.	15s. 7d.
(ii) with dependants ..	23s. 5d.	23s. 5d.

Yours faithfully,

(Signed) W. D. SMITH,
Acting Deputy Prices Commissioner.

The Medical Secretary,
British Medical Association (Victorian Branch),
426 Albert Street,
Melbourne, C.1.

CERTIFICATION OF THE INSANE.

IN May, 1946, following an inquiry by a member of the Victorian Branch, the following letter was addressed by the Medical Secretary to Dr. J. Catarinich, Director of Mental Hygiene.

10th May, 1946.

Section 26 of the *Lunacy Act*, 1928 debars medical practitioners standing in certain relationships from certifying persons as insane. In view, however, of the fact that in several towns in Victoria all the doctors in the area are partners, either with or without assistants, could you supply me with an answer to the following question:

Can X and Y, who are assistants to a partnership of A, B, C and D, between them legally certify a person as insane, although neither of them could act in association with one of the partners?

Dr. Catarinich referred the matter to the Crown Solicitor. The Crown Solicitor's reply, which is published with his consent, is as follows.

Re Question Arising under Section 26 of the Lunacy Act, 1928.

Section 26 of the *Lunacy Act*, 1928, provides as follows:

"26. (1) A person not found lunatic by inquisition shall not be received into any hospital for the insane or licensed house (save under the provisions herein contained) without a request under the hand of some person according to the form in the Ninth Schedule together with such statement of particulars as is contained in the Seventh Schedule nor without the medical certificates according to the form and containing the particulars required in the Fifth Schedule of two medical practitioners neither of whom shall be in partnership with or be the father son or brother of or an assistant to the other, and each of whom shall separately from the other have personally examined the person to whom such certificates relate not more than seven clear days previously to the reception of such person, and such request as aforesaid may be signed before or after such medical certificates or either of them.

(2) No person found lunatic by inquisition shall be so received save under the said provisions without a request in the form in the Ninth Schedule signed by the committee appointed for such person, and having annexed thereto an office copy of the order appointing such committee, and also such statement of particulars as hereinbefore mentioned.

(3) Every person who receives any person into any hospital for the insane or licensed house save under the provisions herein contained without such request accompanied in each case by the statement and medical certificates as aforesaid shall be guilty of a misdemeanour."

In relation to this provision, I am asked to express an opinion on these facts: "Can X and Y, who are assistants to a partnership of A, B, C and D, between them legally certify a person as insane, although neither of them could act in association with one of the partners?"

I have not before me the contracts existing between the parties, but, assuming the assistants under such contracts cannot be regarded as a partnership of five medical practitioners, then in my opinion such assistants can exercise the powers conferred by the section. However, as a matter of prudence I would have thought that medical practitioners who are directly associated in their work would refrain from acting under the section and act only with some independent practitioner. This clearly is the object of the section.

(Sgd.) FRANK G. MENZIES.

Post-Graduate Work.

MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR OCTOBER.

THE programme for October, 1946, arranged by the Melbourne Permanent Post-Graduate Committee, is as follows.

Gynæcology and Obstetrics Refresher Course.—The last of the present series of special courses for supernumeraries at the Women's Hospital will commence on September 23 and continue till October 12.

Continuous Refresher Course.—Medical and surgical refresher classes for ex-service and service medical officers will be continued throughout the month.

F.R.C.S. Part I Course.—The classes in anatomy and histology and in applied physiology and the principles of pathology, which commence in September at the University of Melbourne, will continue until December.

Courses for the Diplomas.—It is anticipated that courses suitable for candidates for Part II of the diplomas of ophthalmology, laryngology and otology, diagnostic radiology, gynæcology and obstetrics, psychological medicine and anæsthesia will commence in October.

Course at Geelong.—Dr. W. S. Newton will give a demonstration on "Chronic Infections of the Lung" at the Geelong Hospital on October 16 at 8.30 p.m. Those wishing to attend should communicate with Dr. Alan Kidd, 216, High Street, Belmont, Geelong.

Enrolments for Courses.—Enrolments for courses in Melbourne should be made with the Secretary of the Committee, College of Surgeons, Spring Street, C.1. Telephone: JM 1547-8.

Correspondence.

SOME COMPLICATIONS AND SEQUELÆ OF THE PROPHYLACTIC INOCULATIONS.

SIR: I was interested in Dr. Gurry's letter of July 20, 1946, and his theory of sustained anxiety contributing to reaction after inoculations. In view of the second case he reports the following experience may be of interest.

I visit a small neighbouring township once a week, and there a farmer's wife consulted me. Believing her to be suffering from early pernicious anæmia, I started her off on injections of "Anahæmin", until she could make arrangements to have a blood examination. She received almost immediate benefit, and after some six injections looked forward with pleasure and anticipation to the injection.

On the day in question—the sixth injection—she had had an ordinary day, her family having for some time made sure

she did little work. She was not apprehensive in any way. I gave her the injection and she left to do some shopping. In ten minutes she was back with every evidence of an intense serum reaction. Her lips were very swollen, as were her eyelids and hands, the conjunctivæ were injected, there was a scarlet rash running up the back of her neck, and she felt intensely itchy. I sat her down and gave her an injection of adrenaline in oil, the only adrenaline I had with me. Before it had time to act she collapsed, and, with her husband's aid, I laid her on a couch. She came around fairly quickly, and after some brandy and water said she felt quite all right. The serum reaction began to fade, and shortly afterwards disappeared. Nine hours later she developed a further attack of intense itching, which cleared up with an injection of "Calceo-Ostelin", and she has been well since.

I quote her case, because I can't feel that prolonged anxiety covers all the picture: there must be a property in the "Anahæmin" itself which occasionally produces hypersensitivity—which is worth remembering.

Yours, etc.,

EWEN B. TUNBRIDGE, M.B., B.S. (Melb.).

Margaret River,
Western Australia,
August 5, 1946.

THE BASIS OF POST-OPERATIVE TREATMENT IN APPENDICITIS AND PERITONITIS: FOLK-LORE OR PHYSIOLOGY?

SIR: It was with surprise and disappointment that I read the excellent article by Mr. Kinsella on appendicitis and peritonitis in the journal of August 10, 1946—disappointment that a life-saving measure was not universally practised. Dr. W. R. Groves had a similar article in THE MEDICAL JOURNAL OF AUSTRALIA of February 19, 1927, and I would suggest that those who have access to the files should read the two articles together.

I think we in Kyneton have been accustomed to using more morphine than Mr. Kinsella suggests. So that my standing orders in actual or threatened peritonitis have been:

1. Patient to have one-quarter or one-sixth of a grain of morphine whenever he is in pain, or is restless, or is mentally bright. The object is to abolish peristalsis.
2. Continuous rectal drip saline solution with a "head" of nine inches of water. It is seldom that, at this slow rate, it is returned.
3. And (quite recently) "Sulphacetamid" ("Solutox" brand) one gramme in four cubic centimetres of water followed by only sufficient water to wash it down, as even water may start gastric peristalsis.
4. No food or fluid by mouth.
5. Every effort to be made to avoid peristalsis (for example, screens around the bed at meal times).
6. If the fluid intake is not satisfactory, intravenous glucose-saline is used.

It is difficult to get nurses to carry out (1) satisfactorily, and a rudimentary knowledge of physics helps them to perform (2), but constant watching on the part of the surgeon is essential.

I apply the same principle to pneumonia with pleurisy. Once the diagnosis is made, harm is done to the patient by allowing unnecessary pain to knock him out, and morphine "splints" the lung somewhat as it does the intestine.

I am sure Mr. Kinsella will have no objection to my calling attention to an article of nearly twenty years ago. The principles are identical; the trains of thought both interesting

Yours, etc.,

J. HORACE DOWNING, M.B., B.S., M.Sc.

Kyneton,
Victoria,
August 13, 1946.

A METHOD FOR THE REMOVAL OF TONSILS AND ADENOIDS UNDER LOCAL ANÆSTHESIA WITH THE PATIENT IN THE RECUMBENT POSITION.

SIR: Dr. Bettington's letter in the issue of August 3, 1946, led me to read his previous article on June 22, 1946, which I had missed.

I would like to inform Dr. Bettington that Dr. H. W. Savige, of Brisbane, removed my tonsils by dissection, under local anæsthesia, whilst I was in a comfortable recumbent position, in 1937. It is a method he has developed and practised since about 1932.

The technique is simple, no gag is used, and the entire procedure in his hands usually takes a bare fifteen minutes.

I have myself used Dr. Savige's technique since 1942. The patient suffers a minimum of discomfort, much less than is incurred from the induction and recovery from a general anæsthetic. The method is very popular with theatre and ward staff, in that very little preparation or after-care is necessary other than confinement to bed for several days.

Yours, etc.,

ALAN PITT.

566, Logan Road,
Brisbane, S.E.2,
Queensland.
August 8, 1946.

SIR: *Re* doing tonsillectomies under a local anæsthetic in the recumbent position.

In reply to Dr. Bettington's query as to when I first adopted the recumbent, or to be more accurate, the Boyle Davis's position of doing tonsils under a local anæsthetic, using the Boyle Davis's gag.

On looking through my records I found that I did the first one at Newcastle Hospital in October, 1938, and I used it continuously until going to the Middle East in November, 1940, when I saw Dr. Ashley Davy using the same position, that is, the Boyle Davis's position, but in place of the Boyle Davis's gag he was using the tongue spatula to retract the tongue.

I considered at that time, and I still consider, that his method was a vast improvement on the use of the Boyle Davis's gag, and since then have abandoned the Boyle Davis's gag, and have used the tongue spatula in its place.

I read Dr. Bettington's article with great interest, but consider the main point is the patient's position, that is, the Boyle Davis's position with the head low, and that a better exposure is obtained by dropping the end of the table one notch.

The advantages of this method are greater to those who have used the Boyle Davis's gag, and the Boyle Davis's position for doing their tonsils under a general anæsthetic, as exactly the same technique for the actual operation is used in both cases.

In 1938 it occurred to me that as one obtained such a good exposure in cases under a gas anæsthesia, it would be an ideal position for a local anæsthesia, and on trying it out I was delighted with the result. This case was, to the best of my knowledge, the first that was tried, but I am always open to correction.

Yours, etc.,

D. G. PERRETT.

Bolton Street,
Newcastle,
New South Wales.
August 13, 1946.

THE ADMINISTRATION OF SYNTHETIC ŒSTROGEN.

SIR: Notwithstanding repeated warnings against excessive administrations of synthetic œstrogens, the practice still goes on.

Time and again patients come along complaining of excessive periods, or when after the menopause, of bloody discharge varying in amount and incidence. In either case, and particularly the latter, one has to decide whether the bleeding is due to the œstrogen or to some associated pathological state. It may seem simple enough to stop the treatment and see what happens, when the problem would be solved by the result.

If the symptoms cease within a reasonable time it may be assumed that the œstrogen was the causal factor, but it is hard to dogmatize what is a reasonable time, and still the anxiety is there that an endometrial cancer may be missed, and during this time of expectancy one imagines the cancer progressing. Frequently, therefore, we feel impelled to perform a diagnostic curettage, and often enough this is unsatisfactory, particularly when the uterus is small and atrophic, and smallness of itself by no means excludes cancer in the post-menopausal cases. There has been a good deal of discussion as to whether cancer is ever caused by œstrogens; consensus of opinion is rather against this possibility. In the last year, however, I have seen two patients develop cancer consequent on the administration of large doses of stilbœstrol, but, of course, on that I cannot say that stilbœstrol was the actual causal factor. Still it is an added anxiety sufficient to increase one's wariness in its use.

If any class of specialist or general practitioner seems least at fault, it may be the general surgeon, who is not so lavish and free with it.

The drug companies seem bent on producing brighter and better œstrogens, no doubt in sympathy with our atomic times, but the main object is to find by trial and error the minimal dosage required for the different threshold of each individual and for the condition for which the stilbœstrol is being given.

In all cases the patient should be warned of the possibilities, as it helps to save repercussions which are sometimes violent.

Yours, etc.,

135, Macquarie Street, Sydney,
August 14, 1946.

RUPERT FURBER.

SPINAL ANÆSTHESIA AND CHLOROFORM: A COMPARISON OF MORTALITY.

SIR: In my article of April 20 I made an attempt, an honest attempt, to find the over-all mortality of spinal anæsthesia, all cases, all methods, all administrants, and to compare this with the results of chloroform anæsthesia, all cases, all methods, all administrants. It revealed an over-all mortality for spinal anæsthesia that was at least twice that of chloroform, and I took also the trouble to point out how fallacious reasoning could obscure the truth. Nowhere did I say that spinal anæsthesia should never be used, and nowhere did I say that chloroform should never be used. That would be to give an opinion, whereas I was looking for the facts. Readers could form their own opinions, but they ought to know the facts. Not that I had no opinions. It happens that I had written my opinion for another purpose, and for another publication, and I wrote it before my article was printed. An opinion may be wrong, but facts are dry things and remain always facts. And this was what I wrote: "I will not say that spinal injections should never be used, but that if they are used, it should be done with at least the same care and circumspection that is counselled for the use of chloroform."

Facts are always facts, and it seems to me that the wrong thing was done by my critics. They were surprised. So was I when I found what the facts were. But they reacted emotionally, without stopping to think. To them the truth could not be true, whatever the evidence. It must be denied, covered up, buried, discredited. And I must be reviled. I must be a ridiculous person, a fool, nay more, a villain, fit for a bad place. Well, I hope not, but let it pass. It is emotional stuff, and it does not hurt.

But the facts have been doing their work, and as reason comes back, we get the comment that ought to have been made at first. This is Dr. Marshall's: "Spinal analgesia is no plaything for tiros." And so say I.

All that emotion was wasted.

Yours, etc.,

175, Macquarie Street, Sydney,
August 19, 1946.

C. E. CORLETTE.

[This correspondence is now closed.—EDITOR.]

DIAGNOSIS OF ANXIETY NEUROSIS.

SIR: The letter in the journal of August 10, 1946, by Dr. A. A. Pain dealing with "anxiety neurosis" is most timely, but I would like to comment on two points.

First among the very real symptoms he details is a most important omission—the fear of impending insanity. This is in my experience so constant and significant that I should base a diagnosis of anxiety neurosis on this alone.

Next as to the need for physical examination. There is one disease which mimics the anxiety state most horribly, and that is pulmonary tuberculosis. The use of mass radiography among troops has been of immense value in picking up such people; even without it—among ex-soldiers—I have been shocked by the number of men who have been treated perhaps for years as anxiety states and who were finally revealed as tuberculous. *Spes phthisica* is in my opinion a late manifestation.

But even among the diseases which he classes as psychosomatic there is the nasty pitfall of the case which has passed beyond the anxiety state into the organic. I have seen one ex-soldier who had been diagnosed (and I consider from a study of his papers, very justly) as an abdominal neurosis—two months after his discharge his duodenal ulcer perforated—operation showed a long-standing ulcer, so scarred that later a gastroenterostomy was needed.

Quite recently a man who had been very vigorously treated for what was deemed an anxiety state by a very competent physician and psychiatrist has returned with an unmistakable thyreotoxicosis, yielding now to full treatment.

Again, is Dr. Pain justified in regarding coronary disease as a result of anxiety neurosis? I have been watching these anxiety state cardiac cases now for over twenty-five years and have been impressed by the very small number who have progressed—or shall we say become transformed—into cases of coronary disease.

The rest of Dr. Pain's letter I consider extremely good clinical common sense.

Yours, etc.,

Craigston,
217, Wickham Terrace,
Brisbane.
August 12, 1946.

S. F. McDONALD.

THE NATIONAL HEALTH SERVICE BILL OF GREAT BRITAIN.

SIR: Dr. Siedlecky in her original letter asserted that every citizen had a "right to free medical service".

I asked her a simple question as to where this alleged right had been derived; although she has written a long letter in reply, she has not answered my question. It is obvious that she cannot do so, as no such right exists. There are certain rights in society, and these are derived from certain fundamental propositions, but the "right" to health is not one of them.

I am glad Dr. Siedlecky realizes that the idea of a "free" medical service is not possible. In a previous correspondence I animadverted on the loose type of thinking and the erroneous use of words that are so common with people of socialistic ideas.

Dr. Siedlecky's original letter is an example of this loose use of words. Right action can only follow upon right thought; erroneous thinking will be followed by erroneous action. Writers on serious subjects should, therefore, be very careful in the use of words and terms that they employ. A "free" medical service can only be instituted and implemented by a high increase in the taxation of wealth and thus further impoverish the producers of wealth. Taxation is a power to destroy, and we are witnesses here in Australia today of its destructive effects.

The health of the people will not and cannot be increased by increased taxation; rather would a reduction in taxation tend to better health—it would mean cheaper and better houses, good clothing *et cetera*.

Dr. Siedlecky's letter of August 3, 1946, contains much loose thought. It might appeal to the emotions, but certainly not to reason. However, she has hit on a very apt expression when she states that medical men should act as "dispensers of health". I am afraid if they ever all work for the Government, that is just what they will become—dispensers.

Yours, etc.,

111, Collins Street, Melbourne,
August 6, 1946.

PAUL G. DANE.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Waterworth, David Henry, M.B., B.S., 1940 (Univ. Melbourne), 113th Australian General Hospital, Concord.

Campbell, Colin Alexander Kemp, Provisional Registration 1946 (Univ. Sydney), 8, Beach Road, Edgecliff.

Clarke, Bruce Gibson, M.B., B.S., 1943 (Univ. Sydney), 15 Camp Hospital, Cowra.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Austin, John Charles, Provisional Registration 1946 (Univ. Sydney), 53A, Raglan Street, Mosman.

Billington, Brian Price, Provisional Registration 1946 (Univ. Sydney), 7, Amarna Parade, Roseville.

Bleasel, Kevin Fabian, Provisional Registration 1946 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Brathwaite, Patricia Constance, Provisional Registration 1946 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

Buckley, Edward Bligh, M.B., B.S., 1940 (Univ. Sydney), Box 26, Post Office, Narrabri.
Cowlishaw, John Abbott, Provisional Registration 1946 (Univ. Sydney), 47, Arnold Street, Killara.
Fairbairn, Jean Margaret, M.B., B.S., 1943 (Univ. Sydney), 4, "Piper House", Longworth Avenue, Point Piper.
Farrar, John Frederick, Provisional Registration 1946 (Univ. Sydney), 21, Princes Avenue, Vaucluse.
Fennell, Thomas Joseph, M.B., 1939 (Univ. Sydney), Flat 3, 7, Shadforth Street, Mosman.
Hicks, Zoe Isobel, M.B., B.S., 1945 (Univ. Sydney), 41, O'Connor Street, Haberfield.
Hilton-Smith, Thea, Provisional Registration 1946 (Univ. Sydney), Balmain and District Hospital, Booth Street, Balmain.
Hollinshead, Frank Mathieson, M.B., B.S., 1943 (Univ. Sydney), Maitland Street, Bingara.
Hytten, Frank Eyvind, Provisional Registration 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
King, Keith John McInnis, M.B., B.S., 1941 (Univ. Sydney), 34, Fort Street, Petersham.
Macintosh, Laurel Jean, Provisional Registration 1946 (Univ. Sydney), Tweed District Hospital, Murwillumbah.
McGlynn, Adrian George, M.B., B.S., 1937 (Univ. Sydney), 300, Campbell Parade, Bondi.
McLaren, William Wotherspoon, M.B., B.S., 1940 (Univ. Sydney), 53, Edward Road, Wahroonga.
Mayer, Loris Lucy, M.B., B.S., 1944 (Univ. Sydney), Dubbo Base Hospital, Dubbo.
Morgan, David Ross, M.B., B.S., 1942 (Univ. Sydney), Mental Hospital, Orange.
Moxham, Ross Moorcroft, M.B., B.S., 1945 (Univ. Sydney), 8, Wiyella Court, West Esplanade, Manly.
O'Connell, Brian Patrick, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Pirie, Bruce Carlyle, M.B., B.S., 1939 (Univ. Sydney), 23, Speed Street, Liverpool.
Potts, Gordon Cameron, M.B., B.S., 1941 (Univ. Sydney), Dubbo Base Hospital, Dubbo.
Rosenthal, Curt, M.B., B.S., 1946 (Univ. Sydney), 128B, Brighton Boulevarde, North Bondi.
Silberstern, Ernest, M.B., B.S., 1946 (Univ. Sydney), Waterfall Sanatorium, Waterfall.
Tonkin, John Richard, M.B., B.S., 1941 (Univ. Sydney), 34, Moseley Street, Strathfield.
Williams, Geoffrey Athol, Provisional Registration 1946 (Univ. Sydney), District Hospital, Lithgow.

Obituary.

JOHN À BECKETT DARVALL BARTON.

WE regret to announce the death of Dr. John à Beckett Darvall Barton, which occurred on August 24, 1946, at Double Bay, New South Wales.

Books Received.

"Pharmacology, Materia Medica and Therapeutics (R. Ghosh's Materia Medica and Therapeutics)", by Birendra Nath Ghosh, M.B.E., F.R.F.P. and S. (Glasgow), F.R.S. (Edinburgh); Seventeenth Edition; 1946. Calcutta: Hilton and Company; Sydney: Angus and Robertson Limited. 8½" × 5¼", pp. 830, with illustrations. Price: 21s.

"Practical Anæsthetics for Students, Hospital Residents and Practitioners", by J. Ross Mackenzie, M.D., D.A. (R.C.P. and S., England), with a foreword by W. C. Wilson, M.B., Ch.B., F.R.C.S.E.; Second Edition; 1946. London: Baillière, Tindall and Cox. 8½" × 5½", pp. 184, with many illustrations. Price: 10s. 6d.

Medical Appointments.

Dr. T. Ambrose has been appointed medical officer of health, Brookton Road Board, Western Australia, in pursuance of the provisions of *The Health Act*, 1911-1944.

In pursuance of the provisions of the *Medical Practitioners Act*, 1938-1945, of New South Wales, the undermentioned have been appointed as members of the New South Wales Medical Board for a period of seven years: Dr. H. R. G. Poate (President), Dr. H. Ritchie, Dr. W. C. B. Harvey, Dr. G. Moncrieff Barron, Dr. H. R. R. Grieve, Dr. J. J. W. Flynn, Dr. C. J. M. Walters, Dr. J. R. Ryan (nominated by the New South Wales Branch of the British Medical Association) and Dr. C. G. McDonald (nominated by the Senate of the University of Sydney).

Dr. R. J. Hoy has been appointed government medical officer at Goulburn, New South Wales.

Diary for the Month.

SEPT. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 4.—Western Australian Branch, B.M.A.: Council Meeting.
SEPT. 4.—Victorian Branch, B.M.A.: Branch Meeting.
SEPT. 5.—South Australian Branch, B.M.A.: Council Meeting.
SEPT. 5.—New South Wales Branch, B.M.A.: Special Groups Committee.
SEPT. 6.—Queensland Branch, B.M.A.: Branch Meeting (Jackson Lecture).
SEPT. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 235, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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